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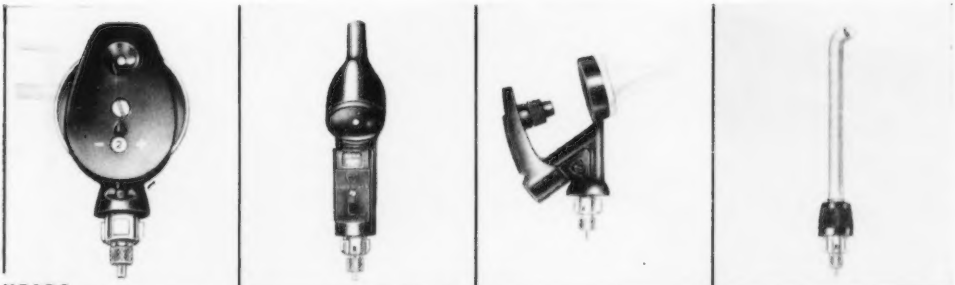

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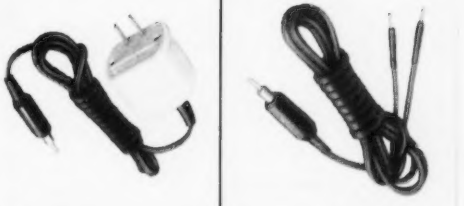

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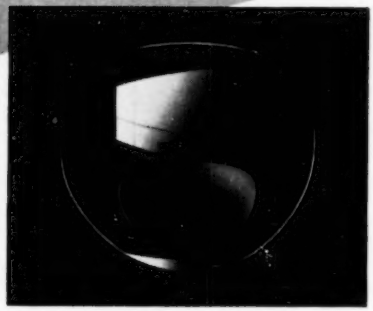

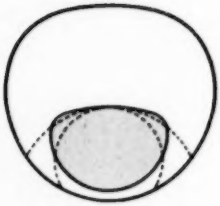

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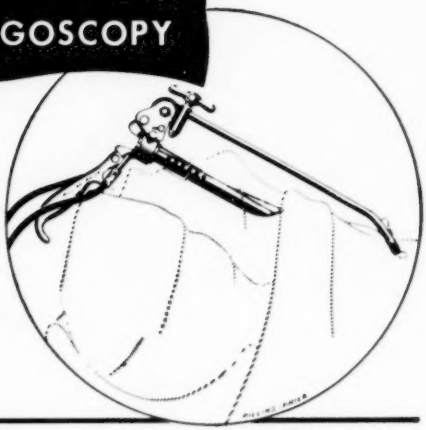

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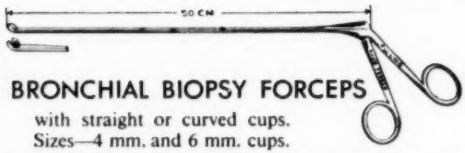

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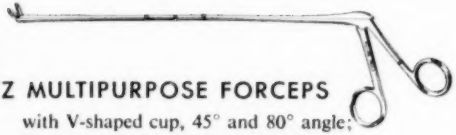

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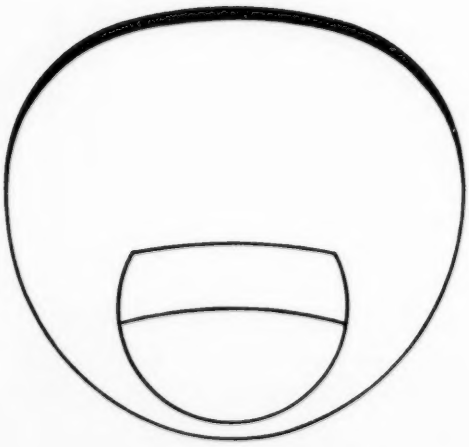

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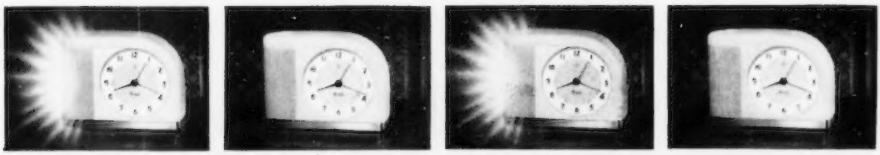

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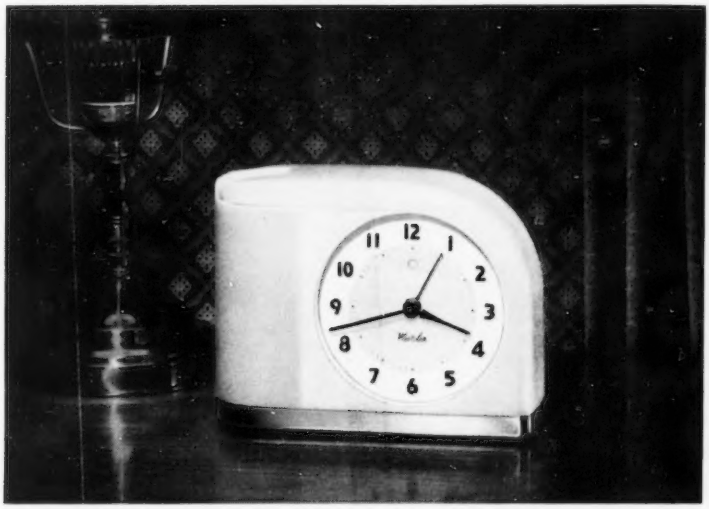

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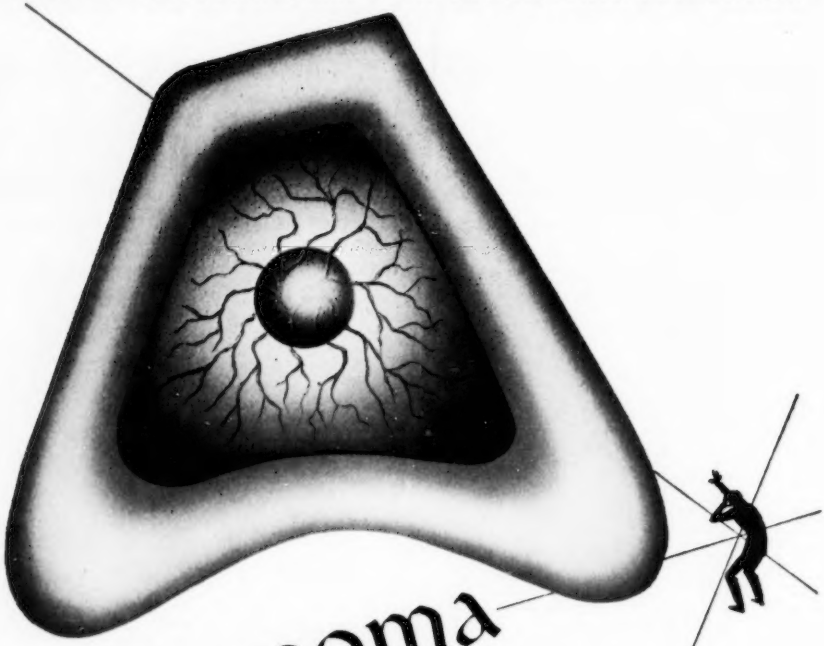

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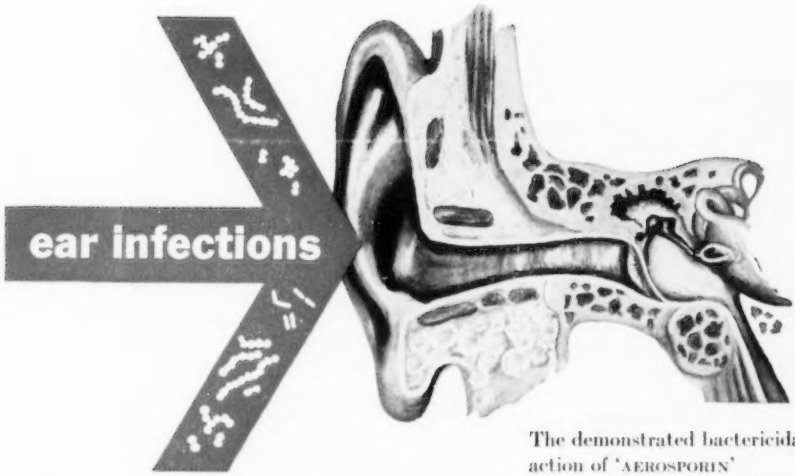

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References: 1. Salvin, S.B. and Lewis, M.L.: External Otitis with Additional Studies on the Genus Pseudomonas, J. Bact. *51*:495, 1946.

2. Hayes, M.B. and Hall, C.F.: The Management of Otogenic Infection, Tr. Am. Acad. Ophth. *51*:149, 1947.

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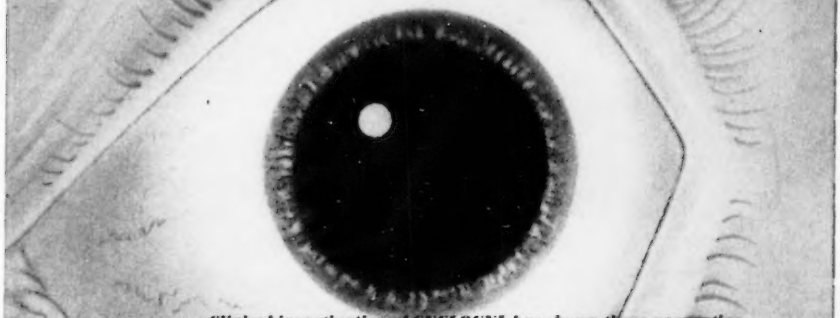

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1. Getter, R. C., and Leopold, I. H.: Arch of Ophth. 49:24 (Jan.) 1953
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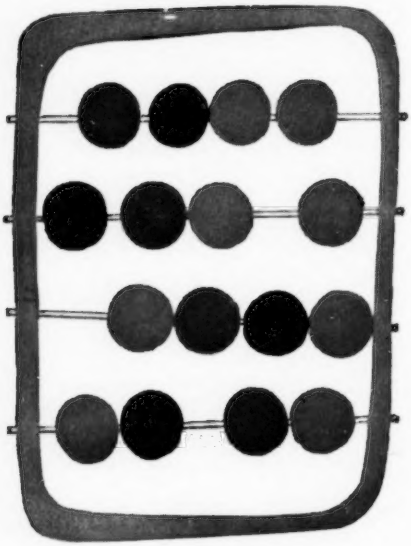

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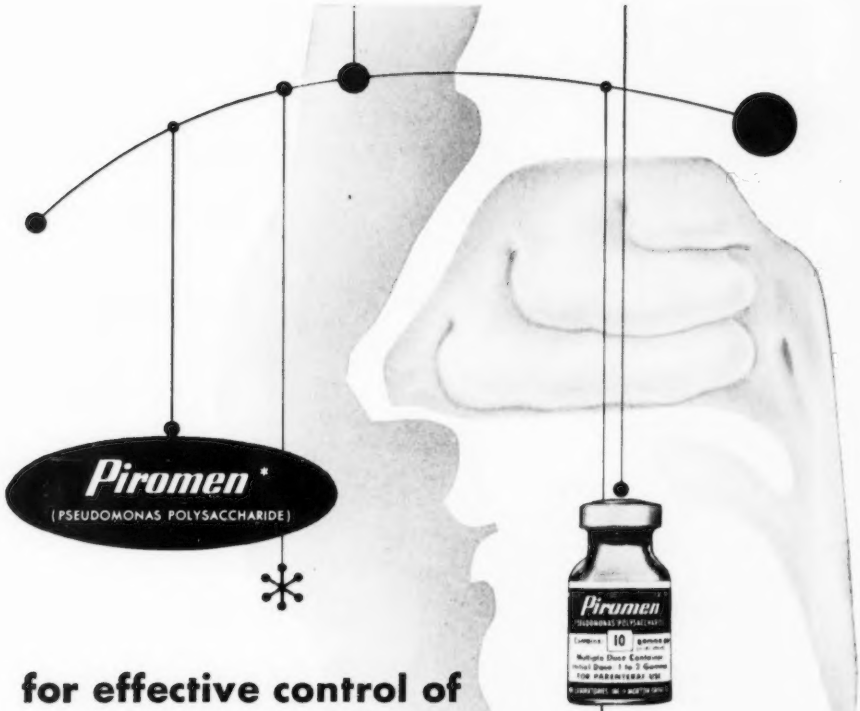

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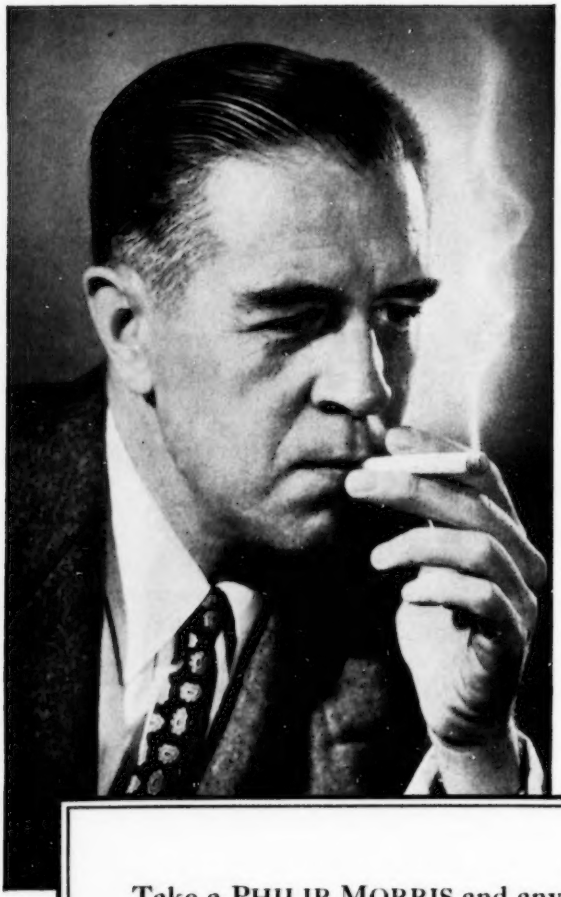

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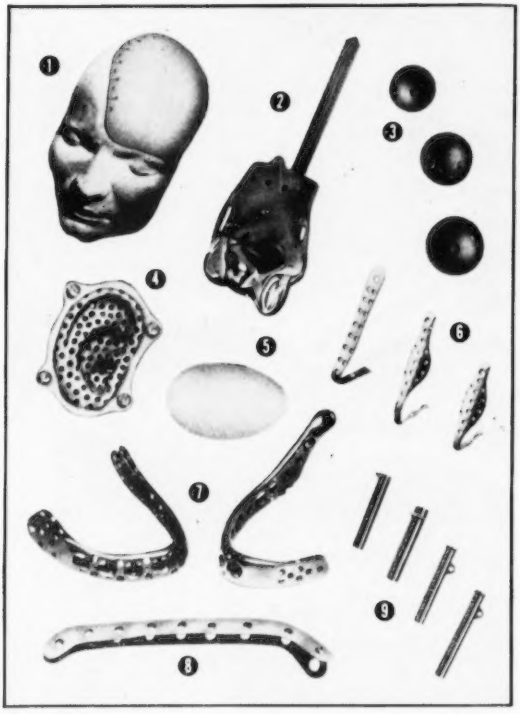

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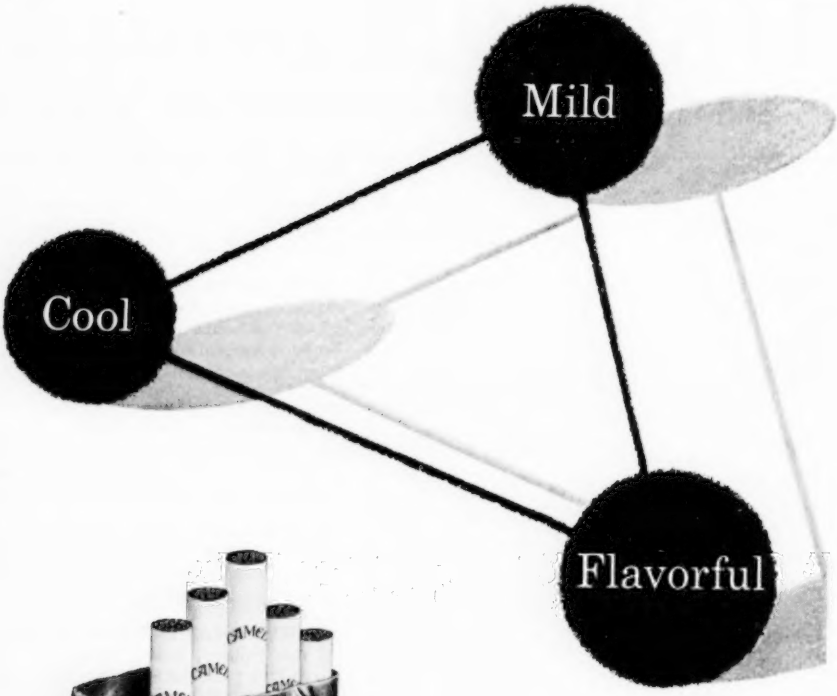

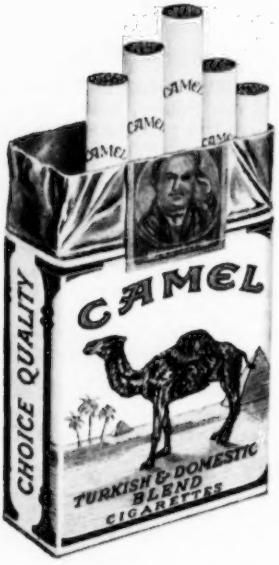

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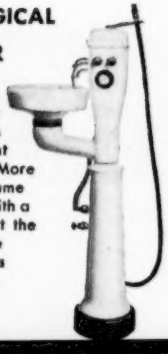

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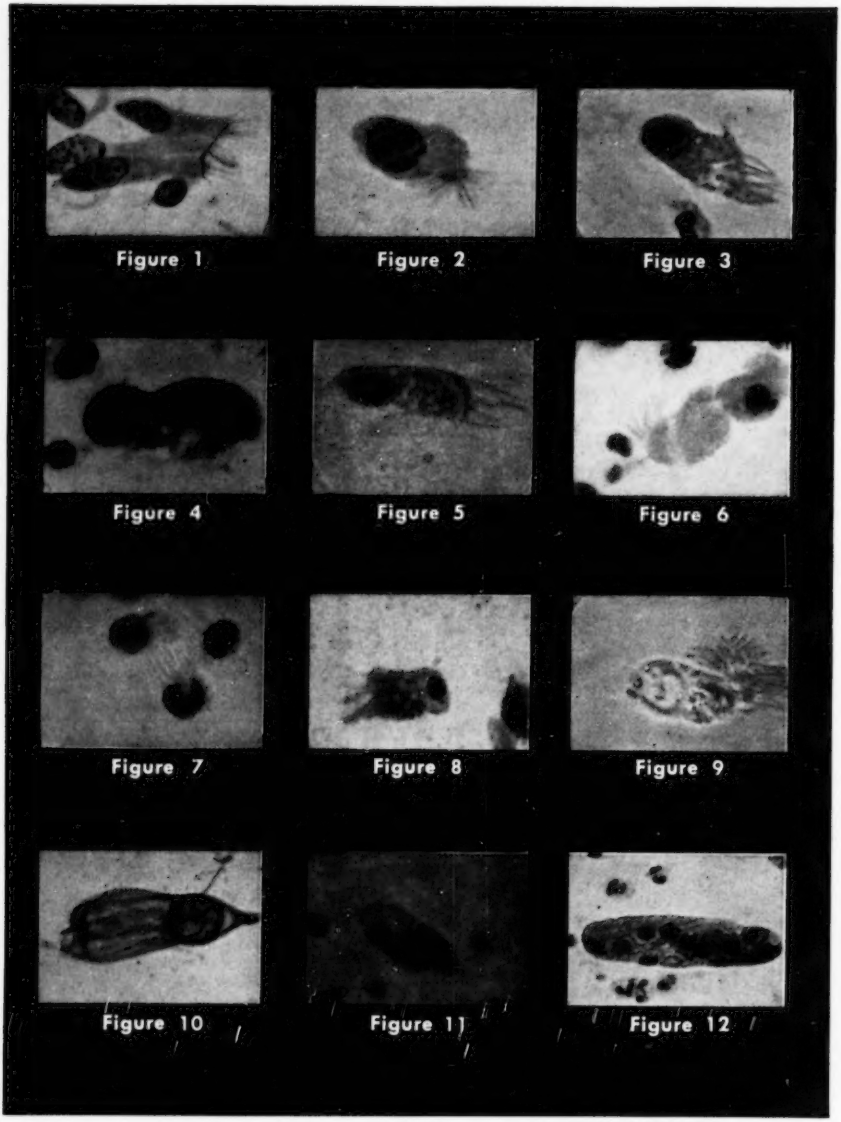

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5

Figure 6

Figure 7

Figure 8

Figure 9

Figure 10

Figure 11

Figure 12

STRUCTURAL CHANGES IN THE CILIATED EPITHELIAL CELLS DURING THE COMMON COLD

WILLIAM T. K. BRYAN, M.D.

and

MARIAN PFINGSTEN BRYAN

BY INVITATION

ST. LOUIS, MO.

THE COMMON cold is still one of the major problems in medicine. One of the greatest difficulties in studies of the prevention of the common cold has been accurate diagnosis.[1,16] Too often patients and physicians confuse allergic reaction with the true infectious cold, now generally believed to be due to Dochez's filtrable virus.[7,8,17] A simple practical and reliable laboratory test for diagnosing the common cold is needed as well as more detailed cytologic observations. This report concerns such a test and study. By using the Papanicolaou fixative and stain,[14,15] with minor variations, we have observed a characteristic pattern of nuclear and cytoplasmic degeneration in the ciliated epithelial cells in the nasal secretions of patients with the common cold.[2,3] This pattern was not found by us in other conditions, such as allergic reactions

Presented at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

LEGENDS FOR COLORED PLATES

The photographs were made from human nasal secretions prepared according to the Papanicolaou technic with the exceptions of the cell stained with Wright's stain in figure 8 and that of the living unstained cell in figure 9. Figures 2 through 9 are from the common cold and represent a series of degenerative changes. Figure 10 is from nasal allergy. Figures 11 and 12 are from the coryza of measles.

FIG. 1—Normal ciliated epithelial cells showing oval nuclei with nucleoli, finely granular chromatin structure and homogeneous cytoplasm. The cilia and ciliary bodies are prominent.

FIG. 2—Early stage of degeneration in a ciliated cell from the common cold. There is margination and clumping of the basophilic chromatin. The pink staining intranuclear bodies appear to be attached to the chromatin.

FIG. 3—Ciliated cell showing slightly more advanced stage of degeneration. The cytoplasm is coarsely granular and the chromatin masses are larger. The pink intranuclear inclusion body is prominent.

FIG. 4—Degenerating exfoliated ciliated cell showing several pink intranuclear inclusion bodies within the halo.

FIG. 5—Advanced stage of degeneration in a ciliated cell with completely pyknotic nucleus, halo, perinuclear bodies and large pink globule in cytoplasm. Some cilia appear on the side of the cell.

FIG. 6—Late stage of degeneration of a ciliated cell showing constriction of the cytoplasm before final breaking apart.

FIG. 7—Two cytoplasmic ciliated tufts. These are numerous during the peak of exfoliation in the common cold.

FIG. 8—A degenerating ciliated cell stained with Wright's stain exhibiting characteristics similar to those stained with the Papanicolaou technic.

FIG. 9—An unstained living ciliated cell from a cold showing the same features. Dark field illumination.

FIG. 10—Columnar epithelial cell wiped from an allergic nose which shows marked edema and vacuolization of the cytoplasm. The nuclear membrane is thickened, outside of which on one side is a large vacuole. The cilia are degenerated.

FIG. 11—Columnar ciliated cell from measles showing streaking of the basophilic chromatin in the double nucleus. Pink globular bodies are scattered in the cytoplasm.

FIG. 12—A multinucleated giant cell from the coryza of measles with coarsely granular acidophilic cytoplasm and cartwheel appearance of the chromatin.

that often simulate the common cold, radiation reactions or subacute and chronic sinus infections. We believe that the pattern of degenerative change may be pathognomonic of virus etiology[4,5] and can be used as a reliable laboratory test for the common cold.

The fact that the nasal mucosa is damaged and that the ciliated epithelial cells are shed during the common cold was well described by the monumental work of Hilding.[11-13] He found desquamated ciliated cells in fresh unstained nasal secretions and showed movies of some of the cells. Biopsy material was also obtained from human volunteers and the pathologic changes in the tissues during successive stages of infection were carefully studied. His search for a bacterial agent which caused the injury to the nasal mucosa was very intensive. It was not found in one hundred tissue scrapings nor in the tissue sections, even though special stains were used. In spite of not finding visible bacteria, the damage to the mucous membrane was evident, along with an infiltration and edema of the submucosa. Our observations fit in with, and supplement, his accurate descriptions of the tissue changes.

Our observations also augment and modify somewhat the careful cytologic studies reported by Hansel.[9,10] Through the years he has emphasized the importance of the eosinophilic-neutrophilic ratio in the differentiation of infections from allergic conditions of the nose. All of us who do nasal cytology recognize the significance of this point. However, from our studies of consecutive smears, we have reason to state that there is a period during the common cold when the smears often show so many eosinophils that at this time the condition cannot be readily distinguished from allergic rhinitis by the eosinophilic-neutrophilic ratio. On the other hand, in pure allergic rhinitis, the degenerative process that we invariably observed in the ciliated epithelium in infectious colds is not found.

Our method consisted of a detailed study of consecutive smears taken twice daily during the course of acute upper respiratory infections. The secretion was blown directly from the nose on to a glass slide or collected in wax paper and spread thinly on the slide with a metal applicator. It is very important to place the smears immediately, before drying, into the fixative of Papanicolaou which consists of equal parts of 95 per cent alcohol and ether. The staining procedure is a special adaptation for smears of the hematoxylin and eosin stains used for tissue sections. Duplicates of these smears were made in Wright's stain for comparison and to aid in identification of the cells. At this date there are thirty-five series and many isolated smears studied whenever they were available.

One complete series of slides, sixty in number, was made from a case of pure ragweed pollenosis. There were numerous other smears from allergic cases with and without infection. Nasal secretions from 12 cases of measles were also observed. The cytology of malignancies and radiation changes in the field of ear, nose and throat have been studied by us with this technic for three years, thus making it possible to have comparative observations.

With the Papanicolaou stain it is possible to accurately differentiate the ciliated cells from the basal and squamous epithelial cells. This stain is not as satisfactory for eosinophils as is the Wright, or the Hansel stain, but the brilliance of the eosinophils in the Papanicolaou stain can be increased by staining one half minute in 0.5 per cent phloxine (prepared in 95 per cent alcohol), following the regular routine after the special dye EA36. We stain nasal smears in the hematoxylin only one to

one and a half minutes rather than six minutes and use only one dip in 0.5 per cent hydrochloric acid rather than six dips as recommended. These are the only variations in the technic and best results are obtained if all of the steps are strictly followed.

The photographs were taken with a Leica camera on a "sliding copying attachment" supported over the microscope. The lenses and the extension tubes were varied to give the desired magnification and size. An oil immersion objective and a 15× ocular lens were used. The magnification on the film can be estimated by comparison with an erythrocyte or a leukocyte. When measured by a stage micrometer it varies from five hundred times to twelve hundred times. A special light meter was calibrated for use on the ground glass.

The pattern of degeneration, which we believe is diagnostic of the common cold, can best be observed in the colored plates.

First are shown a group of normal ciliated columnar cells taken from the human nose and prepared according to the technic of Papanicolaou.[15] In the normal nose these cells are not exfoliated in any number and since there is usually very little secretion, the material was obtained by gentle wiping with a small cotton applicator preferably slightly moistened in saline. Certain distinguishing points in the normal cell are noteworthy. The cilia are seen distinctly attached to the broad end of the cell. The ciliary bodies are often discrete in the pink border. The oval nucleus has a definite thin membrane and shows a fine dispersion of chromatin with two or three nucleoli. It is usually located at the narrow end of the cell opposite the cilia. The borders of the cell may or may not be distinct. The cytoplasm is finely granular and at times appears quite elongated giving the appearance of having marked elasticity. A cytoplasmic tail is usually prominent.

In sharp contrast to the cells obtained from the normal nose are the early degenerative changes that appear in nasal secretion blown from the nose during the first two days of a typical common cold. The chromatin, normally finely granular, is seen in larger aggregates which tend to marginate to the nuclear membrane. This appears to be the first stage of pyknosis, the degenerative process in which the chromatin turns into a solid round mass. The ciliated cells in this early stage appear to be somewhat shortened. A small mass of pink substance is seen within the nucleus among the clumps of chromatin, which with careful focusing may appear to be in contact with a mass of chromatin. As the aggregates of chromatin agglutinate and enlarge, clear areas form, and when the pyknosis is complete, there is a definite halo surrounding the solid ball of chromatin. Often present within the halo are small black and pink bodies. The nuclear membrane becomes quite prominent. The nucleoli disappear. The acidophilic cytoplasm becomes increasingly coarsely granular, shortened and widened. Pink smooth globules, small to large in size, single to several in number, tend to appear in the cytoplasm near the nucleus. The cilia remain intact and in some instances they grow from the sides of the cells.

As the degeneration progresses after the second day, the cytoplasm tends to separate from the nucleus by elongating slightly and constricting near the nucleus. Finally the two portions break apart. The process is interesting to observe in the living condition. Throughout this obvious degeneration the cilia remain intact, even after the nuclear portion breaks off. Many pyknotic nuclei with clear halo, often containing one or more of the small perinuclear bodies, are seen separated from the cy-

toplasm which still retains the tuft of cilia. Hilding[11] described the tufts of cilia in his studies. In our fresh preparations the beating of tufts of cilia without nucleus has been observed for as long as two hours.

At the same time in these pictures some of the ciliated cells, which have not broken apart, exhibit "empty nuclei." These seem to have lost from the cell the black mass of chromatin. A clear area is present without visible structure. The striking thing is that in a single slide during the peak of exfoliation all manner of nuclear and cytoplasmic material is seen scattered unattached over the slide. This includes whole pyknotic nuclei, that is, the spherical black masses with or without halo and small perinuclear black bodies, as well as cytoplasmic fragments with tufts of cilia. Sometimes the black pyknotic nuclear fragments are seen to be phagocytosed by monocytes.

The peak of the exfoliation is from the second to the fifth day of the infection. The actual number of ciliated cells varies extensively from case to case and with the severity of the nasal symptoms. The exfoliated ciliated cells never approach the number of neutrophils in the secretion. During the peak of exfoliation one may see two to four in a single oil immersion field. The later stages of degeneration predominate after the second day. The cellular fragments are then seen in large numbers. After the first week of an acute upper respiratory infection the degenerating ciliated cells are difficult to find. They decrease rapidly after the fifth day and have not been noted after the twelfth day. They are completely gone from the secretions before the neutrophils and other cells disappear. They have not been seen in pus irrigated from the sinuses in subacute and chronic conditions. The general cytologic picture varies individually with the severity and duration of symptoms but this specific pattern of the ciliated cells has been especially constant in patients with the common cold. This same pattern was observed in the nasal secretions from several cases in which the clinical diagnosis was influenza.

In cases of pure pollen allergy the specific pattern has not been noted. Occasional ciliated cells that are exfoliated may show normal nuclei with boggy swollen vacuolated cytoplasm. Others may show vacuolation within and around the nuclei as well as in the cytoplasm. By wiping the mucosa many epithelial cells are obtained among which goblet cells are seen. Cilia are not usually distinctly visible on these swollen vacuolated cells. The nuclei frequently show vesiculation but this pattern is very different from the nuclear and cytoplasmic changes characteristic of the ciliated cells in the common cold. In allergic cases which are complicated by an acute infectious cold the specific degenerative pattern of the ciliated cells may be found. It is believed that this distinguishing factor may be helpful in differential clinical diagnosis.

The coryza of measles was similarly studied by smears according to the technic of Papanicolaou[15] and certain distinctive cytologic features were found. Most striking were the giant cells heretofore described only in surgical and autopsy material from measles. Warthin[19] fully described these cells in the tonsil and in the pharyngeal tissues. Others[18] have reported them in the laryngeal, bronchial and esophageal mucosa as well as in the appendix, spleen, thymus, liver and lymph glands. They are large multinucleated cells easily seen in our nasal smears from measles. We have also observed that the nuclei of both the columnar and basal epithelial cells in measles appear to degenerate in a pattern somewhat resembling a cartwheel. Cilia are not frequently found. These features are certainly different

from the changes described in the common cold.

Other types of cellular degeneration and necrosis do not resemble the degenerating pattern of the ciliated cells seen in the common cold. The abnormal nuclei of malignant cells are very different. In two cases of malignancy of the nasal pharynx it was interesting to note that when the patients had a cold, the characteristic degenerated ciliated cells with inclusion bodies were found in the secretion along with the tumor cells. Malignant cells after exposure to roentgen rays show marked degenerative changes with multiple nuclei, vesiculation, intracellular swelling, bizarre shapes and even pyknotic nuclei, but these alone or in combination do not simulate the pattern of degeneration seen in the common cold.

To satisfy ourselves that the morphologic details observed in the ciliated cells in the common cold were not artefacts, we examined from the same material, fresh unstained wet smears, as well as those prepared with both the Wright and Papanicolaou stains. If the smears were very lightly stained with Wright's stain after being air-dried, not flamed, and within one half hour after being made, the same degenerating ciliated cells could be seen. They probably would not have been recognized except for the fact that they were first clearly noted in the Papanicolaou stain. The cilia are not well preserved but can be recognized. The halo and inclusion bodies which are of a different shade from the pyknotic nucleus may be verified. It is also very interesting to note that in the fresh unstained preparations the same structural variations can be observed when the lighting is indirect.

DISCUSSION

What is the significance of this type of degeneration with inclusion bodies, as observed in the ciliated cells? A possible answer is found in the statement by Cowdry[6], an authority on virus diseases.

> When the following features are noted in a section it is likely that a virus has been at work.
>
> 1. A considerable number of inclusion-laden nuclei which can be arranged in a series representing stages in development. This indicates an active process in which the nuclei exhibiting the most advanced alterations were affected first and the others in succession.
> 2. A change in which the accumulation of acidophilic material forming the inclusion, is accompanied by margination of basophilic chromatin on the nuclear membrane, a disappearance of nucleoli and ultimate death and disintegration of the cells. This suggests that the inclusion formation is not merely an intranuclear heaping up of material effected without injury.
> 3. A cellular reaction characterized by hyperplasia, hypertrophy or necrosis.

Our material from the evidence thus far observed fulfills these requirements as stated by Cowdry.

This statement, we wish to bring out, is not proposed as proof that these inclusion bodies are actual virus elements, but rather, that the present evidence would identify the inclusion bodies with changes which occur in cells infected with virus. Dr. E. V. Cowdry, Dr. Walter Siebert, pathologist, and Dr. Walter P. Covell have studied our slides and concur in this opinion.

Even though the cytologic diagnosis of the common cold may rest on the finding of this special degeneration of the ciliated cells, the study of the whole general cytologic picture is also of significance in each case for a better understanding of acute upper respiratory infections as well as other nasal conditions.

SUMMARY

1. Nasal cytology in various conditions has been studied by using the

Papanicolaou technic by which intracellular detail and cilia are clearly demonstrated.

2. A specific pattern of structural changes occurring in the exfoliated ciliated epithelial cells during acute upper respiratory infections has been observed which we believe is diagnostic of virus activity in these cells.

3. The method is offered as a simple reliable laboratory test for the diagnosis of the common cold.

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DISCUSSION

GEORGE E. SHAMBAUGH, JR., M.D., Chicago, Ill.: The Bryans have given us a diagnostic test for the common cold that should prove valuable in future attempts to solve our No. 1 public health problem. The common cold is the commonest disease of man, averaging in the United States alone over 300,000,000 cases a year.

We know quite a bit about the common cold. From the fine work of Dochez we know that it is a highly contagious disease due to a specific virus transmitted mainly by air-borne droplets during the first twenty-four hours of symptoms, with an incubation period of eight to forty-eight hours in the unfortunate victim who inhales the droplets. From the beautiful work of Hilding we know that the tissue attacked by the virus is primarily the ciliated epithelium of the respiratory tract which is so badly damaged that much of it becomes necrotic and sloughs. Damaged ciliated cells appear in untold thousands in the nasal secretions during the first two or three days of the

disease. So great is this sloughing of ciliated cells that in some areas only a single layer of cuboidal cells remains on the basement membrane. Regeneration of the ciliated cells begins almost at once, with a return to normal in about two weeks.

The temporary loss of the wonderfully efficient protective mechanism of cilia and mucous blanket allows the streptococci and pneumococci and other bacteria lurking always in the nasopharynx to invade the tissues with a resulting sinusitis, otitis media, bronchitis or pneumonia that is far more debilitating and dangerous than the original virus infection.

Today we have efficient drugs against the secondary invaders and complications. We have yet to find an effective means of prevention or treatment of the primary virus infection of the ciliated epithelium. In a nutshell the problem is to prevent the infectious droplets from reaching the susceptible individual, or to immunize the individual against the specific virus, or to find an effective drug that will destroy the virus before the virus destroys the ciliated epithelium.

In any research into the prevention or treatment of the common cold it is necessary to differentiate sharply between the true virus infection and an allergic reaction. This differentiation is not always easy for the common cold and the allergic reaction produce similar symptoms. When a patient tells us that he catches cold every morning or every few days or every week or two, it is a good guess that he has an allergy, probably to house dust. When a patient has only one to three colds a year, with a free interval of several months between each one, it is a good guess that he has the true virus infection. But for scientific research, guesses are not enough, nor can we always rely on the finding of eosinophils in the nasal smear, for both Hilding and the Bryans have observed a brief stage in the common cold where eosinophils occur in the tissues and nasal secretions in increased numbers. That is about the third day of the cold.

Hitherto the only certain way of diagnosing the true virus infection common cold was by monkey inoculation and transmission according to the method of Dochez. Now for the first time we have a simple practical and apparently reliable laboratory test. This test is based on identification in the nasal smear stained by the method of Papanicolaou of the characteristic changes in the ciliated cells produced by the cold virus. With this stain the changes produced by the virus are easily differentiated from changes due to trauma, radiation or allergy. I believe that this contribution of Dr. and Mrs. Bryan is an important one in the fight against public health enemy No. 1.

ALLERGIC MANAGEMENT OF SOME COMMON EAR CONDITIONS

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ANY discussion of allergic disease is helped by a definition of allergy as the term is used by the author. We have attempted to limit our diagnosis of allergic ear disease to one in which an antigen-antibody reaction occurs, requiring specific therapy for control.

The three tissues most susceptible to allergic disease are the skin, the gastrointestinal mucosa and the respiratory mucosa, since they are the barriers to invasion of the human organism by pathogenic organisms. Accordingly these tissues possess to the highest degree the ability to elaborate specific antibodies against foreign invaders. In certain persons with an inherited predisposition these tissues also elaborate antibodies against ordinarily harmless foods or inhalants. When such an individual inhales or eats the substance to which antibodies have been produced a reaction occurs in his tissues due to the union of antigen and antibody, with tissue damage, liberation of histamine, edema, eosinophilia, smooth muscle spasm and increased flow of mucus from the mucous glands. This is known as the allergic reaction, and hay fever is the classical example in the respiratory mucosa.

In the field of otorhinolaryngology there is now general acceptance and recognition of the prevalence of allergic nasal and sinus disease. Allergy of the middle ear should not be surprising, for the eustachian tube and middle ear are lined by the same respiratory mucosa that so readily develops clinical allergies. Allergic reaction of the skin of the external auditory canal and auricle is also easily understandable. Less obvious is allergy of the inner ear until we reflect that the endolymphatic labyrinth is a derivative of skin, coming from the ectodermal otocyst.

In the past five years the writings of Koch,[7] Ashley,[1] Jones et al.,[4] Jordan,[5] Kuhn[8] and others have given impetus to an increasing awareness of allergic states in the ear. In particular, statistical studies of the percentage of occurrence of allergy in an otologic practice, such as made by Jordan[6] in 1952, are enlightening. We feel that confirmation of or comparison with such studies is worth while.

The diagnosis of allergic disease is complicated by the fact that there is no single accepted conclusive test to prove an allergy. It is unfortunate that patients in general and a surprising number of physicians have come to accept the skin test as infallible evidence for or against an allergic disorder. Reliance upon the skin test is very often misleading and disappointing in our experience. Of much greater value is the history, and the finding of increased eosinophils in secretions or tissues is strong presumptive evidence of an allergic reaction. The most reliable proof of an allergic etiology and of a specific allergen lies in the therapeutic test: the relief of

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symptoms by removal of a substance or by treatment with an extract of the substance, and the recurrence of symptoms upon re-exposure to the substance or upon stopping treatment. Thus, long-standing chronic diseases of the ear which have resisted all other forms of treatment and which respond to allergic management should be considered as allergic in origin.

Applying the criterion of the therapeutic test to 993 new otologic cases seen in our practice in one year from May 1951 until May 1952, 115 cases (11.6 per cent) were considered to be allergic in origin.

THE EXTERNAL EAR

External otitis is a common otologic problem. Recent studies by Singer et al.[11] and Senturia[10] have established the prime importance of the Pseudomonas group and other specific micro-organisms, as well as the relative unimportance of the fungi, in the etiology of chronic external otitis. Such chronic cases generally respond to thorough cleansing and appropriate local medication. Certain cases of external otitis fail to respond to local measures, and eventually specific allergic management supplies the solution. We diagnose these cases as allergic eczema, finding such variable allergens to be responsible as drugs, cosmetic contacts, foods and inhalants including dust, feathers and pollens. Six new cases of allergic external otitis were found in the one year period previously mentioned. An illustrative case follows.

Case 1. Lt. D.S., aged 28, was seen because of a four months' duration of itching and discharge from both ears. The symptoms had been persistent in spite of many local medications, as well as several environmental and climatic changes due to Army travel. Eventually a history of eating chocolate fudge and popcorn almost nightly, with intradermal skin tests yielding large positive reactions to corn, chocolate, coffee, spinach and cucumbers (fig. 1) dictated an elimination diet study. Corn was the main suspect at first, but chocolate was finally proved by repeated clinical tests to be the primary offender. This patient, who was inordinately fond of chocolate, found that he could expect itching and drainage from the ears within four hours after eating chocolate, and that by the next day the discharge would cease.

We have also observed that following the fenestration operation a specific allergy to an inhalant, a food, a cosmetic or sometimes to a local medication is not infrequently responsible for a persistent discharge from the operated cavity. This occurred in the following case.

Case 2. Mrs. E. C., aged 45, was successfully fenestrated in June 1950 with hearing restored and maintained well above the practical level. The cavity healed uneventfully and was observed to be perfectly dry and clean one year after surgery. In July 1951, this completely healed cavity began to itch and to drain a foul, tan-colored purulent secretion. Despite frequent cleaning and various local medications the drainage continued. The history of the onset of symptoms in July, after an environmental change to the patient's summer home, raised the suspicion of an allergic external otitis due to summertime atmospheric molds or pollens, offenders we have detected in other cases. This clue was entirely misleading, and a subsequent trial of treatment with nonspecific agents such as staphylococcus toxoid and the fungus mixture used by Hansel[3] also resulted in failure. In desperation, a basic elimination diet followed by specific feeding tests according to Rinkel's technic[9] resulted in a pronounced flare-up in itching and drainage accompanied by hives on the left side of the face and neck immediately following the wheat feeding. Wheat omission has healed the cavity. On two subsequent occasions following wheat ingestion the discharge recurred for a brief period.

THE MIDDLE EAR

Allergy of the middle ear is encountered much more frequently than allergy of the outer or inner ears. Of the 115 cases of aural allergy seen in one year, 40 were diagnosed as allergic chronic suppurative otitis media with a

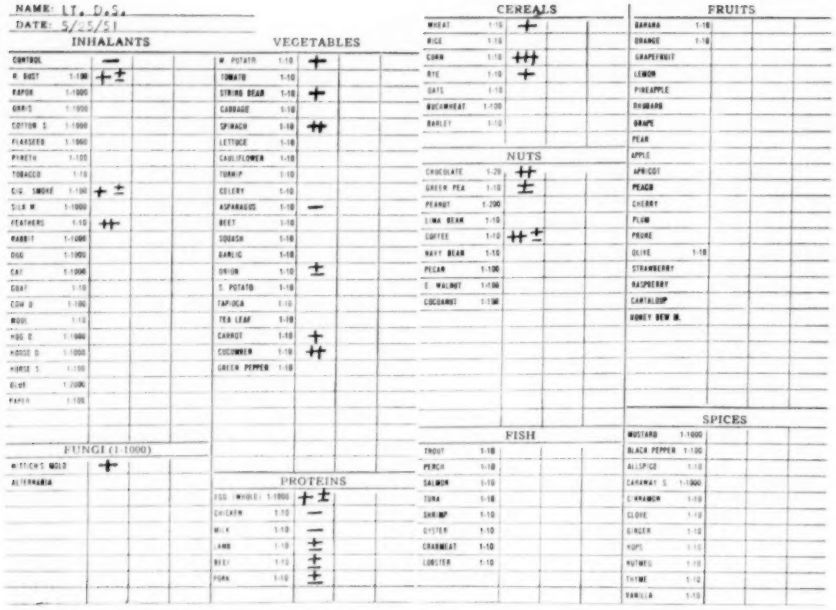
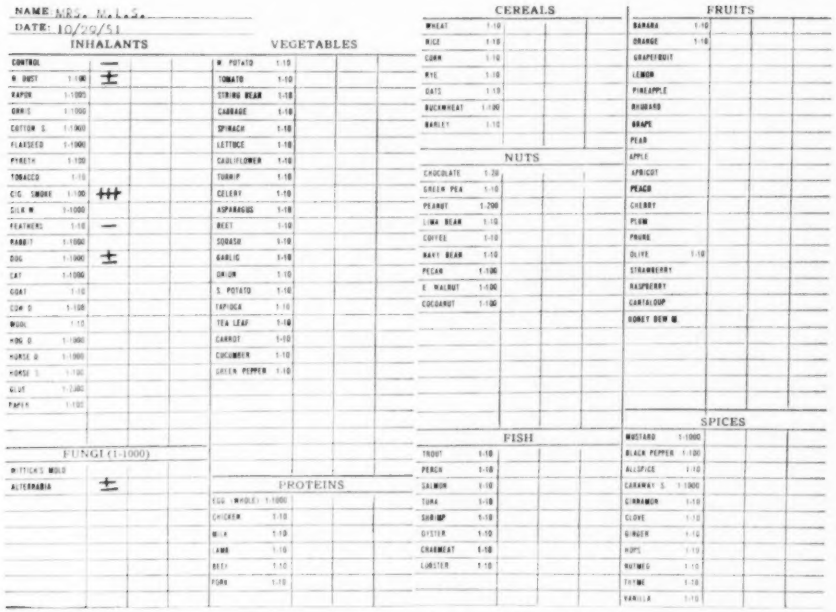


FIG. 1.—Intradermal skin tests for selected inhalants and for the "common foods" which occurred most often in the diet of the patient, Lt. D.S. Chocolate was the only food of the strong skin reactors to be clinically important.

central type of perforation. The characteristic finding in such cases is usually a profuse, thin mucoid or thicker, gelatinous secretion containing a varying number of eosinophils. These cases resist therapy as a rule until the allergic factor is controlled. One of us in a recent study of a technic for closure of long-standing central perforations of the tympanic membrane found that in 129 of these cases with chronic suppurative otitis 46 were allergic.

Allergic involvement of the mucosa of the eustachian tube and of the middle ear can also produce the picture of repeated attacks of acute otitis media, as it did in 6 patients of the 115. Or an allergic reaction in the mucosa of the tube can result in chronic secretory otitis media, as occurred in 41 cases of the 115. We believe that in every case of chronic secretory otitis media that is not due to an obvious obstruction of the eustachian tube by tumor or adenoid, and that resists the usual treatment of inflations, paracentesis, and radium or roentgen irradiation of the nasopharynx, an underlying allergy should be searched for. If there is also a chronic nasal allergy, the likelihood of an allergic etiology is enhanced. The excellent response of chronic or recurrent secretory otitis media to allergic management is particularly gratifying in children, with the reasonable expectation of preventing the cases of irreversible chronic adhesive otitis and acquired attic cholesteatoma which are the all too frequent sequelae of long-standing chronic secretory otitis media. Jordan,[6] in particular, emphasizes this point.

The following three cases illustrate the three types of middle ear allergy.


NAME MRS. M.L.S.
DATE: 10/29/51

INHALANTS

CONTROL		—
H. DUST	1:100	±
KAPOK	1:1000	
ORRIS	1:1000	
COTTON S.	1:1000	
FLAXSEED	1:1000	
FIREFH	1:100	
TOBACCO	1:10	
CIG. SMOKE	1:100	+++
SILK H.	1:1000	
FEATHERS	1:10	—
RABBIT	1:1000	
DOG	1:1000	±
CAT	1:1000	
GOAT	1:10	
COW D.	1:100	
WOOL	1:10	
HOG D.	1:1000	
HORSE D.	1:1000	
HORSE S.	1:100	
GLUE	1:1000	
PAPER	1:100	

FUNGI (1:1000)

PITTICK'S MOLD	
ALTERNARIA	±

VEGETABLES

W. POTATO	1:10
TOMATO	1:10
STRING BEAN	1:10
CABBAGE	1:10
SPINACH	1:10
LETTUCE	1:10
CAULIFLOWER	1:10
TURNIP	1:10
CELERY	1:10
ASPARAGUS	1:10
BEET	1:10
SODASH	1:10
GARLIC	1:10
ONION	1:10
S. POTATO	1:10
TAPIOCA	1:10
TEA LEAF	1:10
CARROT	1:10
CUCUMBER	1:10
GREEN PEPPER	1:10

PROTEINS

EGG (WHOLE)	1:1000
CHICKEN	1:10
MILK	1:10
LAMB	1:10
BEEF	1:10
PORK	1:10

CEREALS

WHEAT	1:10
RICE	1:10
CORN	1:10
RYE	1:10
OATS	1:10
BUCKWHEAT	1:100
BARLEY	1:10

NUTS

CHOCOLATE	1:20
GREEN PEA	1:10
PEANUT	1:200
LIMA BEAN	1:10
COFFEE	1:10
NAVY BEAN	1:10
PECAN	1:100
E. WALNUT	1:100
COCOANUT	1:100

FISH

TROUT	1:10
PERCH	1:10
SALMON	1:10
TUNA	1:10
SHRIMP	1:10
OYSTER	1:10
CRABMEAT	1:10
LOBSTER	1:10

FRUITS

BANANA	1:10
ORANGE	1:10
GRAPEFRUIT	
LEMON	
PINEAPPLE	
RHUBARB	
GRAPE	
PEAR	
APPLE	
APRICOT	
PEACH	
CHERRY	
PLUM	
PRUNE	
OLIVE	1:10
STRAWBERRY	
RASPBERRY	
CANTALOUP	
HONEY DEW M.	

SPICES

MUSTARD	1:1000
BLACK PEPPER	1:100
ALLSPICE	1:10
CARAWAY S.	1:1000
CINNAMON	1:10
CLOVE	1:10
GINGER	1:10
HOPS	1:10
NUTMEG	1:10
THYME	1:10
VANILLA	1:10

FIG. 2—Borderline reaction to intradermal skin test for house dust which was a poor diagnostic aid in comparison with the clinical history.

Case 3. Mrs. M.L.S., aged 36, complained of an intermittent right otorrhea "all of her life." A thorough history elicited the pattern of almost constant aural discharge associated with persistent nasal blocking, postnasal discharge and severe nightly coughing, following the first headcold in the fall and lasting until the following summer. Previous local treatments and various antibiotics had failed to influence the nasal and ear symptoms. Despite a doubtful result from an intradermal skin test for house dust (fig. 2), the excellent "dust history" warranted a trial on house dust injections. The mucoid ear secretion dried quickly on dust therapy, and the best relief of both the ear and the respiratory symptoms resulted from 0.2 cc. of 1:5000 dilution of Endo dust. This was the first time the otorrhea had ever been cleared up by treatment during the winter time. Closure of the central perforation in the right ear was carried out, with restoration of hearing to a near normal level (fig. 3).

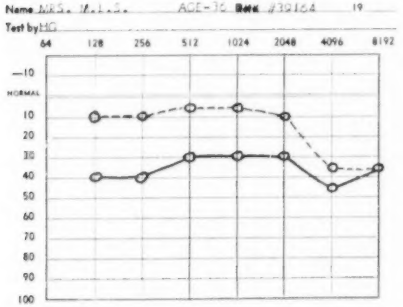


FIG. 3—O—O represents a hearing gain of 23 decibels in the right ear after successful control of an allergic chronic otitis media by dust hyposensitization followed by closure of the 3 x 3½ mm. central perforation.

Case 4. Master R.V.W., aged 8½, complained of impaired hearing and repeated attacks of acute otitis media in both ears since early childhood. A chronic nasal blocking with thick mucoid discharge suggested a nasal allergy. This was confirmed by findings of eosinophils in the nasal smear. Despite negative intradermal skin tests, a pronounced sensitivity to egg and house dust was demon-

strated by clinical trial. Both factors had to be controlled to achieve a complete clearing of the nasal symptoms and marked improvement in the hearing for the first time in many years (fig. 4), and a marked reduction in the frequency and severity of the attacks of acute otitis media. Previous tonsillectomy and adenoidectomy at the age of 2 years and again at 4 years, and roentgen therapy to the nasopharynx and eustachian tubes at the age of 6½ years had failed to influence the ears at all.

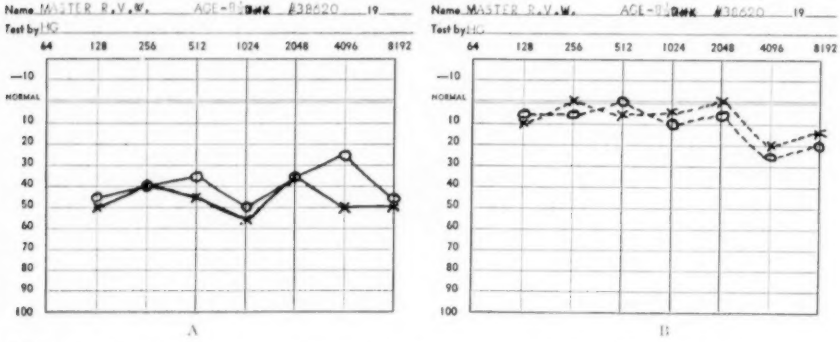


FIG. 4.—A. O——O Right ear and X——X Left ear —— audiogram prior to any allergic management.
B. O——O Right ear and X——X Left ear —— audiogram following control of nasal and aural allergy.

Case 5. Mrs. I.H., aged 46, complained of a yearly recurrence since childhood of a bilateral hearing impairment, worse in the left ear, with a sense of severe pressure in the ears and an occasional watery drainage. This occurred each winter along with a persistent postnasal discharge and a "hacking" cough, and had not responded to many local nasal treatments and inflations. During a ten-year period of residence in Alabama, the ear symptoms and coughing had been absent. Examination revealed a bilateral secretory otitis media with moderate retraction of the drum membrane and small attic retractions. An initial intradermal skin test dose of 0.01 cc. of 1:1 billion dilution of dust extract was followed by immediate improvement in the cough and hearing. Subsequent maintenance dust therapy with 0.15 cc. of 1:10 million dilution obtained complete relief of the cough and postnasal discharge and of the secretory otitis media without a paracentesis. The successive audiograms (fig. 5) during treatment show a rapid improvement in the typical audiometric curves of secretory otitis with a remarkable lack of permanent hearing damage considering the many years' duration of the condition.

THE INTERNAL EAR

In recent years the condition of endolymphatic hydrops has been recognized and diagnosed with increasing frequency as a cause for certain types of nerve deafness. The majority of cases of endolymphatic hydrops are of unknown origin. Allergy, but not of the antigen-antibody type, is held by Day[2] to be responsible as an intrinsic allergy affecting the internal auditory artery, and by Williams[12] as a physical allergy of the capillary bed either in the stria vascularis or in the branches of the internal auditory artery supplying the cristae and maculae or both together.

In endolymphatic hydrops the patient shows the picture of a low tone perceptive deafness that usually fluctuates widely, at least in the early stages; the symptom of diplacusis can be demonstrated in many cases; the patient complains of a sense of pressure or fullness in the ear, a roaring type of tinnitus and attacks of vertigo. Not all of these symptoms need to be present in any one case to justify the diagnosis of labyrinthine hydrops.

While the majority of cases are of unknown etiology, a very definite minor-

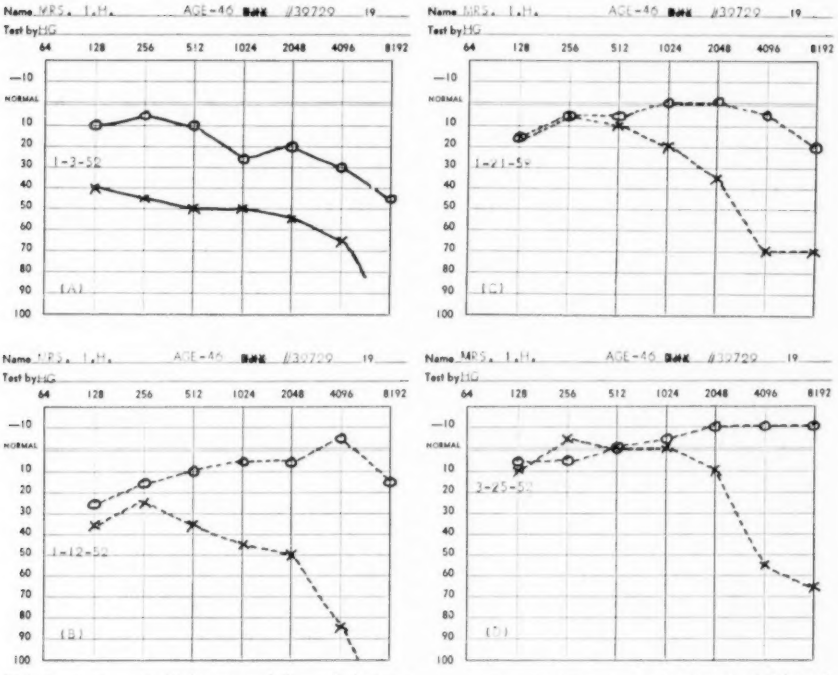


FIG. 5.—A. O——O Right ear and X——X Left ear ... secretory otitis media.

B. O- - -O Right ear and X- - -X Left ear ... and ear symptoms after the intradermal skin test with dilute housedust extract.

C. O- - -O Right ear and X- - -X Left ear ... after first treatment dose of dust.

D. O- - -O Right ear and X- - -X Left ear ... secretory otitis media with patient on maintenance dust therapy.

ity we find are the result of a specific antigen-antibody allergy of the endolymphatic labyrinth to a food or an inhalant. In our series 13 cases were classified as allergic, with 5 cases proved by therapeutic test and the remaining 8 cases listed as probable but still undergoing a trial by allergic work-up. Although this represents but a small fraction of our cases of hydrops, the possibility of an underlying specific allergy should be considered in every case of labyrinthine hydrops. If the patient also has other allergic symptoms such as asthma, allergic rhinitis, dermatitis or gastrointestinal symptoms from certain foods, an allergic etiology for the inner ear symptoms is even more probable.

The diagnosis of the specific allergen in allergic labyrinthine hydrops presents considerable difficulties due to marked spontaneous fluctuations in the symptoms. Thus, the patient who improves on elimination of a food might have improved if nothing had been done. When the patient has other allergic symptoms such as a chronic rhinitis, the discovery of the specific allergen to which he is allergic is greatly facilitated. As in other allergies, the final proof lies in the therapeutic test. The following case brings out a number of points we have emphasized.

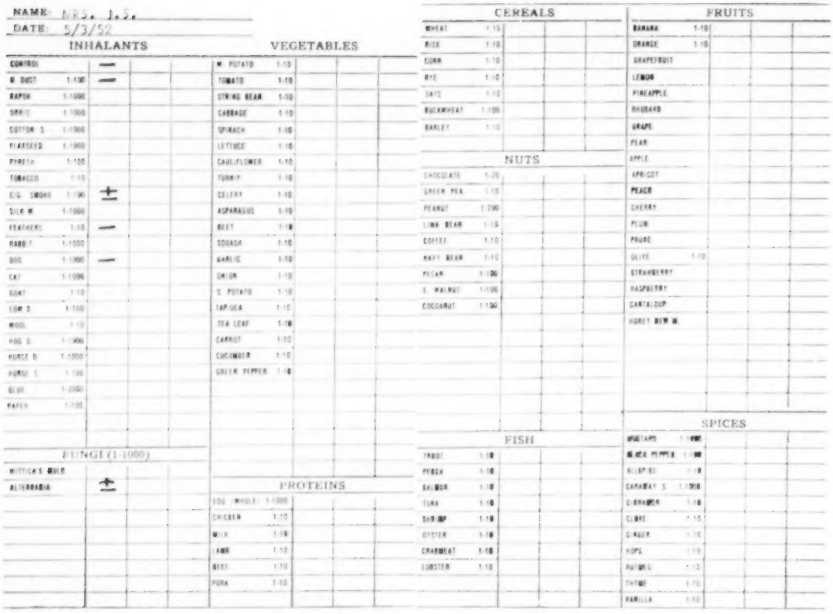


FIG. 6.—Negative intradermal skin test to dog dander in patient, Mrs. J.S., who was clinically sensitive to her own cocker spaniel's dander.

Case 6. Mrs. J.S., aged 31, complained of severe attacks of rotary vertigo with vomiting lasting about 12 hours. The sudden vertigo was always preceded by a day of severe sore throat followed by marked nasal stuffiness and profuse watery nasal drainage. The entire episode was afebrile. There was no complaint of tinnitus or of hearing impairment during or following the attacks, but both ears felt "full and tight" during the attack. The patient felt weak and unsteady for a month following these attacks, which had occurred two or three times yearly for two years since moving from Chicago to a small community in Iowa. There had also been a persistent nonseasonal nasal stuffiness and postnasal discharge since moving.

Examination revealed only a moderately boggy nasal membrane. The audiogram showed normal hearing, and cold caloric testing gave equal and normal responses, with an induced vertigo similar to but much less severe than the patient's attacks. A careful history revealed the only apparent significant change in home furnishings or environment to be a cocker spaniel. Despite a negative intradermal skin test to dog dander (fig. 6), removal of the dog and thorough housecleaning was advised. This was done with clearing of the patient's nasal symptoms and a much more rapid relief from the unsteadiness which had followed the last attack of vertigo. Two subsequent exposures to the dog at the home of the patient's mother were followed by a return of nasal symptoms, a sore throat and the typical rotary vertigo previously experienced. A skin test with an extract from the cocker spaniel's dander would have been interesting in the case of this patient.

CONCLUSIONS

We have found that stubborn cases of external otitis, of secretory otitis media or of chronic suppurative otitis media of the benign type which have resisted all other therapy are frequently due to specific inhalant or food allergy and respond to specific allergic management. The finding of eosinophils in the secretion is helpful in directing our attention to an allergic etiology. The skin tests are of occasional help, but as often

as not they are negative or misleading, so that the proof of the allergic etiology lies in the therapeutic test with dilute dust therapy or elimination of a food or environmental contact.

An allergic etiology for labyrinthine hydrops more often than not cannot be demonstrated. Nevertheless, there is a small but important minority of cases which prove to be definitely allergic to a food or an inhalant, and which will respond to specific allergic management.

The diagnosis of allergy of the ear in our experience is consistent with the diagnosis of chronic allergies of the nose and sinuses; namely, that the history is of greater value and dependability in determining the allergen than are the skin tests.

As in the treatment of chronic nasal allergy, the best results in aural allergy will occur only when the otolaryngologist learns to make his own allergic diagnostic studies and carries out his own allergic management of these cases.

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DISCUSSION

R. E. JORDAN, M.D., Pittsburgh, Pa.: Two years ago a survey was made of the combined practices of Dr. Kenneth Day and myself to determine the percentages of allergic patients in our practice. Seven hundred twenty-seven new otologic patients were seen during 1950, and of these 27 per cent had conditions caused or complicated by allergy. This figure is somewhat higher than the percentage just reported by Dr. Derlacki. The difference lies in the individual concept of the term "allergy." In our survey were included the physical or intrinsic allergies as well as the antigen-antibody reactions. Allergic manifestations resulting from emotional disturbances or glandular imbalances were included. All cases of Ménière's disease with or without vertigo were included in the allergic group, since we believe this condition is due to an autonomic dysfunction or a physical allergy. The antihistaminic drugs are frequently employed as a diagnostic test, and if the results are favorable the patient is considered allergic. It is obvious that a much broader concept of the term "allergy" was used in our survey than in the one just reported.

Of the 727 cases seen in 1950, 111 were diagnosed as chronic secretory otitis media. Of these 97 were found to have an associated allergy. These 111 cases represent 13.3 per cent of our practice. The importance of early diagnosis and control of chronic secretory otitis media cannot be overemphasized. In our practice 8 cases have been observed to progress from a simple secretory otitis media to perforation of Shrapnell's membrane to subsequent attic cholesteatoma. Fixed invagination of Shrapnell's membrane is commonly seen following repeated attacks of secretory otitis media. Inward rupture of Shrapnell's membrane can result in the formation of attic cholesteatoma. This complication may account

for the failure of the antibiotics to reduce the number of radical mastoidectomies for chronic mastoiditis with cholesteatoma.

In our experience, cases of Ménière's disease caused by a specific antigen are rare. Minimal doses of subcutaneous histamine have been the medical treatment of choice. However, complete allergic study is done on all patients, and supplementary allergic management instituted as indicated.

In order to maintain uniform control of cases with Ménière's disease it is necessary to eliminate or control any associated allergies. An example is the patient with Ménière's disease and hay fever. Although the patient may be symptom-free on a histamine regime, labyrinthine symptoms may occur during the hay fever season if the ragweed sensitivity is not controlled.

I wish to congratulate Dr. Derlacki for his excellent factual presentation of the role of allergy in otology.

RUSSELL I. WILLIAMS, M.D., Cheyenne, Wyo.: Dr. Derlacki has very wisely limited his discussion of allergy to the antigen-antibody type of reaction requiring specific therapy for control because of the controversy over the exact physiologic mechanism of allergic manifestations. His case reports are very good examples.

It is true that allergic disease is complicated by the fact that there is no single, accepted, conclusive test to prove an allergy. The individual's history and the examination of the secretions or the tissues are strong presumptive evidences of an allergic reaction. The most reliable evidence is that obtained in a therapeutic test or by specific therapy.

In my experience I have found small doses of Trichophyton and Monilia albicans in conjunction with staphylococcus toxoid to be very beneficial. A thorough cleansing of the ear canals is all that is needed to clear up many of these cases. This treatment in conjunction with the injection therapy has proven very satisfactory in our hands.

By no means do I wish to leave the impression that all cases of external otitis are of allergic origin. However, the possibility should be considered in the diagnostic work-up. It is not a panacea, but I believe you would all be very gratified if you give particular attention to the allergic side of the picture as well as to the other possibilities in your differential diagnosis and treat your patients accordingly.

Allergy of the middle ear occurs more frequently than is commonly accepted. The commonest variety, of course, is the secretory otitis or otitis with effusion. This, in our experience, is one of the most important entities as related to common ear conditions. Secretory otitis is seen quite often with an intercurrent infection and it is necessary to treat both the infection and the allergy to get the desired result.

Endolymphatic hydrops can be proven to be of a definite allergic origin in a small percentage of cases. It is gratifying when we do find such a case; treat it with specific therapy, and it is certainly worth while to continue our search for an allergic etiology. It is agreed that the direct, definite etiologic factor is quite controversial.

Possibly the theory of physical allergy as proposed by Dr. Henry L. Williams would be accepted more universally than that of a true antigen-antibody type of reaction. There is much work yet to be done to determine the etiologic factor of this entity.

I should like, again, to stress the importance of the history. I would like also to stress the importance of the examination of the secretions in making the diagnosis of allergy. We should accept the skin test as an adjunct. The final proof is in the therapeutic test.

From the treatment standpoint the common error in overlooking the state of the individual at the time of the allergic manifestation is not considered to its fullest extent. In this I refer to the autonomic nervous system; and here again is another lead for the study of allergy physiology. For example, we have all seen the occurrence of allergic reaction when an individual comes in contact with a very small amount of an antigen to which he is sensitive and can show varying degrees of allergic manifestation. At other times the same individual is able to tolerate the antigen with impunity and show little or no reaction.

How can the etiologic factor definitely be proven to be of an allergic nature, a specific inhalant or food?

The proof of allergic etiology or specific allergy depends upon a therapeutic test. If the removal of the specific substance or treatment with an extract of this substance relieves the symptoms and if the symptoms recur on re-exposure or after treatment has been discontinued, we may assume that the particular substance is responsible for the symptoms.

I would like to place emphasis on Dr. Derlacki's suggestion that the otorhinolaryngologist should be equipped to make his own allergic diagnostic studies and, furthermore, carry out his own allergic management.

EFFECT OF PROGRESSIVELY BUFFERED SOLUTION OF EPHEDRINE ON THE NASAL MUCOUS MEMBRANE

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CHICAGO, ILL.

NASAL vasoconstrictors occupy a commanding position among the various therapeutic measures employed by the general public for the relief of nasal congestion and the common cold. Ephedrine, the first of the nasal vasoconstrictors to be introduced into clinical practice, is still the most widely used. Its success has led to the synthesis of other nasal vasoconstrictors, some more potent, others less potent than ephedrine. Although ensuing relief is temporary, overcoming the discomfiture caused by intranasal obstruction is welcome to distraught patients. There is little to indicate that the current passion for psychosomatic phraseology will alleviate millions of inflamed and allergic American noses.

For physicians to disregard the opportunity of producing palliation when such effect is easily possible is tantamount to complete therapeutic neglect. Since the function of physicians is to prescribe measures of relief in the event a cure cannot be obtained, habitual rejection of the use of so simple a therapeutic device as a physiologic nasal vasoconstrictor in the treatment of intranasal congestion will often drive patients to over-the-counter purchases and self-medication.

The creation of standards for rational nasal medication has been assisted greatly by cognizance of the histologic and physiologic effect of various nasal preparations on nasal or sinal mucous membrane, ciliary motility, the hydrogen ion concentration of nasal secretion, and by noting the presence or absence of local and systemic side effects. Fantus' dictum[10] that a hydrogen ion concentration near that normal to the mucous membrane is of greater importance for applications to the mucous membranes than the matter of isotonicity has provided a firm foundation for rational, physiologic nasal medication. So far as the pH values of local medicaments are concerned, valid therapeutic conclusions have been drawn in a variety of clinical fields from measurements of the hydrogen ion levels of various secretions found in their original positions on underlying mucous membrane and tissue surfaces.

During the past decade, several communications have appeared calling attention to the local effect of nasal medicaments with various pH values on the nasal mucous membranes of laboratory animals and human subjects. At a time when the introduction of the local use of sulfonamide compounds as prophylactic and curative agents in the treatment of infections of the nasal cavity and paranasal sinuses was being greeted with unrestrained zeal, a note of disillusionment was struck by Fletcher[12] who reported necrosis and charring of the mucous membranes of the maxillary sinus following irrigation of the sinus with highly alkaline 5 per cent and 10 per cent solutions of sodium sulfathiazole over a three-week period. Futch

The author is indebted to Dr. Kurt Stern, Assistant Director, Mount Sinai Medical Research Foundation, for assistance with the histologic study.

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and his associates[13] observed the effect of 5 per cent and 10 per cent solutions of sodium sulfathiazole with an approximate pH of 10 on the nasal mucous membranes of rabbits and found that the 5 per cent solution of sulfathiazole sesquihydrate exerted an early and destructive effect on the nasal mucous membrane. Hunnicutt[18] instilled 0.5 per cent, 1 per cent and 5 per cent solutions of sodium sulfathiazole in the nasal cavities of mice three times a day for fourteen days. Purulent exudate was detected during the first three days in about half the mice. Gundrum[14] studied the effect of 4.7 per cent solutions of sodium sulfathiazole, sodium sulfadiazine, and butanoylsulfanilamide on the nasal mucous membranes of rabbits. Microscopic study of the nasal tissues of the animals treated with sulfanilamide derivatives showed injury to the entire nasal mucous membrane with great damage to the olfactory area. This was more pronounced in animals in which sodium sulfathiazole and butanoylsulfanilamide were used. Sodium sulfadiazine, while not so completely destructive, was definitely injurious. Dickson[3] tested six commercial sulfonamide nasal preparations on the nasal mucous membranes of rabbits and found no injury produced by preparations with pH values ranging from 5.0 to 9.4.

Callomon[2] studied the microscopic tissue changes produced in the nasal mucous membrane of normal mice by prolonged local administration of various sulfonamide compounds bearing different pH values. In varying degree, tissue changes were found during treatment with all the sulfonamide compounds, with pronouced differences of degree in the injury instituted by the preparations. The least irritation was noted in animals given 3 per cent solution of sulfathiazole in propylene glycol (pH 5.1), with a pH value near the normal physiologic range of nasal secretions in man. The greatest injury and a slow return to normal were observed following use of 2 per cent solution of diasone in propylene glycol, which evinced the highest acidity among the solutions used (pH 4.8). One of the commercial preparations, a combination of desoxyephedrine and sodium sulfathiazole in aqueous solution (pH 9.2) caused an inflammatory and partly caustic effect. Finally, Fabricant, Hollender and Anderson[8] conducted studies on rabbits demonstrating that various solutions of sodum and calcium penicillin (containing from 1,000 to 5,000 units per cubic centimeter) applied topically to the nasal mucous membrane of rabbits for prolonged periods have no delecterious effects. The solutions employed had pH 6.0 to 6.5.

METHOD OF STUDY

In this study a single nasal vasoconstrictor preparation was employed. An attempt was made to have all components alike except for progressively buffered differences in pH values. In investigations undertaken by others, it should be pointed out that the various nasal preparations used not only differed in pH values but in chemical structure as well. This introduced elements into the study that made difficult a proper evaluation of the pH factor in nasal medication.

Forty-five rabbits were employed in the present investigation, of which five died during the course of experimentation. The right nasal passages of forty presumably normal rabbits received a progressively buffered solution of 1 per cent ephedrine hydrochloride in physiologic sodium chloride, the left nasal passages serving as a control. The pH ranges of the appropriate nasal solutions were prepared according to the following phosphate systems:

Phosphate systems	*pH range*
KH_2PO_4 H_3PO_4	2.5 to 5.0
KH_2PO_4 Na_2HPO_4	5.2 to 8.0
Na_2HPO_4 Na OH	8.0 to 12.0

Each of the treated animals received three drops of the appropriately buffered isotonic nasal solution from a standard medicine dropper three times daily for a period of thirty days. Two rabbits received the 1 per cent solution of ephedrine hydrochloride with each pH value, which was increased by increments of 0.5 in a range from pH 2.5 to pH 12. Thus two rabbits received a solution with a pH of 2.5, two a solution with a pH of 3.0, two a solution with a pH of 3.5, etc.

Each of the animals was killed at the end of thirty days by means of an intravenous injection of air. After the turbinal structures were removed, sections were prepared for histologic study. Hematoxylin and eosin stains were used.

RESULTS

At autopsy, the treated nasal cavities of nine rabbits revealed varying amounts of gross purulent exudate, while the untreated nasal cavity of one rabbit evinced gross purulent exudate. In the former group, gross purulent exudate was demonstrated in animals treated with solutions of 1 per cent ephedrine hydrochloride buffered to pH 3.0, 3.5, 4.0, 4.5, 7.0 (in both rabbits), 11.5 and 12.0 (in both rabbits). The single rabbit whose untreated nasal cavity showed gross purulent exudate was the one whose treated nasal cavity had received the ephedrine preparation buffered to pH 4.0.

In numerous instances the microscopic sections revealed a similarity between the ephedrine-treated and the untreated nasal mucous membranes, with histologic changes of a minimal character. For the most part, the pathologic changes in the respiratory nasal mucous membranes consisted of slight to considerable hyperemia; acute, subacute, and chronic inflammation; and varying degrees of suppuration. The treated respiratory nasal mucous membrane of thirteen of the forty rabbits showed slight to considerable hyperemia, with the tissue of three displaying additional chronic inflammatory changes and suppurative foci. Varying degrees of acute and subacute inflammation and edema solely were detected in seven rabbits; acute and subacute inflammation with suppuration was seen in three rabbits; acute and chronic inflammation in two rabbits; acute and chronic inflammation with suppuration in three rabbits; chronic inflammation solely in ten rabbits; and chronic inflammation with suppuration in two rabbits. In the untreated nasal cavities of the forty rabbits under study, no appreciable histologic changes were detected in eight animals, while slight to moderate hyperemia was observed solely in fourteen rabbits, and varying degrees of acute and subacute inflammation were found in four rabbits. In the remaining fourteen rabbits, slight to moderate chronic inflammation occurred, with one of the animals showing suppuration.

TABLE I

SUMMARY OF PATHOLOGIC CHANGES IN THE NASAL MUCOUS MEMBRANES OF RABBITS AFTER THIRTY DAYS OF INTRANASAL INSTILLATION OF A PROGRESSIVELY BUFFERED SOLUTION OF 1 PER CENT EPHEDRINE HYDROCHLORIDE IN PHYSIOLOGIC SODIUM CHLORIDE

RABBIT	pH OF SOLUTION	CHANGES IN TREATED NASAL CAVITY	CHANGES IN UNTREATED NASAL CAVITY
1	2.5	Hyperemia	No appreciable change
2	2.5	Hyperemia; numerous goblet cells	Slight hyperemia
3	3.0	Subacute inflammation and edema	Hyperemia
4	3.0	Severe subacute inflammation with gross purulent exudate in nasal cavity	Moderate subacute inflammation and edema
5	3.5	Mixed acute and chronic inflammation with gross purulent exudate in nasal cavity	Moderate hyperemia
6	3.5	Moderate hyperemia	Slight hyperemia
7	4.0	Moderate hyperemia, moderate chronic inflammation, and some edema	Slight hyperemia
8	4.0	Considerable acute and chronic inflammation with gross purulent exudate in nasal cavity	Subacute inflammation with gross purulent exudate in nasal cavity
9	4.5	Considerable acute and chronic inflammation with moderate amount of purulent exudate in nasal cavity	No appreciable change
10	4.5	Slight chronic inflammation	Slight chronic inflammation
11	5.0	Subacute inflammation with moderate edema	Slight chronic inflammation
12	5.0	Subacute inflammation	Slight subacute inflammation
13	5.5	Severe subacute inflammation mixed with chronic inflammation	Slight chronic inflammation
14	5.5	Slight chronic inflammation	Moderate hyperemia
15	6.0	Moderate chronic inflammation	Severe acute inflammation
16	6.0	Moderate subacute inflammation	Slight chronic inflammation
17	6.5	Mixed acute and chronic inflammation	Chronic inflammation
18	6.5	Slight chronic inflammation	Slight chronic inflammation
19	7.0	Considerable acute inflammation with gross purulent exudate in nasal cavity	No appreciable change

TABLE I (Continued)

RABBIT	pH OF SOLUTION	CHANGES IN TREATED NASAL CAVITY	CHANGES IN UNTREATED NASAL CAVITY
20	7.0	Considerable acute purulent inflammation with moderate amount of purulent exudate in nasal cavity	Slight chronic inflammation
21	7.5	Several foci of subacute inflammation	Hyperemia
22	7.5	Slight subacute inflammation	No appreciable change
23	8.0	Considerable hyperemia	Slight hyperemia
24	8.0	Slight hyperemia	No appreciable change
25	8.5	Diffuse chronic inflammation with extensive edema	Slight hyperemia
26	8.5	Subacute inflammation	No appreciable change
27	9.0	Moderate hyperemia and edema	Slight hyperemia
28	9.0	Slight hyperemia	No appreciable change
29	9.5	Moderate hyperemia with focal metaplasia to transitional epithelium	Slight hyperemia
30	9.5	Moderate hyperemia and chronic inflammation	Slight hyperemia
31	10.0	Chronic inflammation	No appreciable change
32	10.0	Hyperemia with some areas of suppuration	Slight hyperemia
33	10.5	Moderate hyperemia and chronic inflammation	Moderate hyperemia
34	10.5	Severe chronic inflammation	Diffuse chronic inflammation
35	11.0	Considerable hyperemia with chronic inflammation	Slight hyperemia with chronic inflammation
36	11.0	Pronounced chronic inflammation	Slight chronic inflammation
37	11.5	Severe chronic inflammation with moderate amount of purulent exudate in nasal cavity	Slight chronic inflammation
38	11.5	Moderate chronic inflammation	Slight chronic inflammation
39	12.0	Numerous foci of suppuration with chronic inflammation and moderate amount of purulent exudate in nasal cavity	Moderate chronic inflammation
40	12.0	Considerable chronic inflammation and hyperemia with small amount of purulent exudate in nasal cavity	Moderate chronic inflammation and hyperemia

COMMENT

In a general way, this study reveals that the nasal mucous membrane of the untreated nasal cavities of rabbits is less susceptible to histologic change than is the mucous membrane of nasal cavities treated over a period of thirty days by a progressively buffered solution of 1 per cent ephedrine hydrochloride in physiologic sodium chloride. Highly acid solutions of 1 per cent ephedrine hydrochloride (pH 3.0; 3.5; 4.0; and 4.5) and highly alkaline solutions of 1 per cent ephedrine hydrochloride (pH 11.5 and 12.0) are more likely to produce traumatic and suppurative mucosal changes than either slightly acid or slightly alkaline preparations. Strangely, the two rabbits subjected to treatment with the nasal preparation buffered to a neutral pH 7.0 both evinced acute inflammation with gross purulent exudate in the treated nasal cavities. Just why this occurred is difficult to explain.

In considering the widespread use of laboratory animals in experimentation involving the nasal administration of drugs, Kelemen and Sargent[19] point out that spontaneous pathologic changes are not always fully appreciated. Such changes duplicate a large variety of conditions ranging from slight inflammation to the most severe destruction, findings that are often claimed to be produced experimentally. This is true both of rats and rabbits. While studying the incidence of spontaneous sinusitis in rabbits, Hilding[17] observed infected paranasal sinuses in otherwise apparently healthy animals; he found that when a rabbit becomes ill or debilitated, rhinitis and sinusitis are prone to develop. From these observations it may be concluded that the frequency of nonexperimental pathologic changes warrants the utmost caution in evaluating findings that have been made by comparisons with conditions in "control" tissues or animals.

As for the nasal mucous membrane of the human nasal cavity, this structure is continuously subjected to the lashings of acute nasal infections, weather conditions, allergens, smoke, dust, and chemical substances. "Normal" respiratory mucous membrane, in the true sense of the term, exists only during the first few months of life, seldom remaining normal because of its vulnerability to inflammatory changes. Consequently, if inhalation of air produces changes in the respiratory mucous membrane of the human nasal cavity, it can come as no surprise that prolonged use of some nasal medicaments produces similar findings in one degree or another at the same site. In this connection, it may be inferred that the nasal mucous membrane of rabbits responds to its respiratory environment in like fashion; hence the occurrence in the present investigation of numerous instances of identical, minimal inflammatory changes in both the ephedrine-treated and the untreated nasal mucous membranes of the rabbit.

A laboratory animal such as the rabbit can generally be considered an important vehicle for evaluating the local effect of nasal preparations on nasal tissues, since it is presumed to possess a mucosa more nearly comparable to that of human beings than the mucosa of any other laboratory animal. Nevertheless, there are occasions when the evidence and the conclusions derived from animal experiments—in this instance, the nasal cavity of the experimental rabbit—are limited in their applicability to the study and treatment of disease in man. Under such circumstances, further evidence should be sought in the human being himself.

pH FACTOR IN HUMAN NASAL PHYSIOLOGY AND BACTERIOLOGY

Disturbances in the human nasal cavity are often an expression of altered

physiologic activity rather than the end result of gross pathologic change. In this connection, one of the more important chemical phenomena involving nasal secretions is pH, hydrogen ion concentration. Under normal conditions the secretions of the nose and the paranasal sinuses maintain a biochemical composition that fluctuates within a limited range according to the demands of physiologic function, while under abnormal or pathologic conditions the biochemical reactions are altered.

The pH of nasal secretion in situ in clinically normal adult nasal passages ranges from approximately 5.5 to 6.5, whereas in infants and young children it ranges from 5.0 to 6.7. During acute rhinitis, acute sinusitis, and during the more active phases of allergic rhinitis, the pH of nasal secretion is found on the alkaline side, and then shifts back to the acid side when the stage of clinical resolution is reached.[6,15] Nasal pH varies with rest and sleep; prolonged rest and sleep promote an acid trend. Nasal pH is more acid during the night than during the day.

The relationship of the hydrogen ion concentration of nasal secretion to cellular reactions has been commented on independently by Tweedie[20] and Buhrmester,[1] who found that patients with pH values of 6.5 or below always had negative cultures. Tweedie made the observation that with elevation of the pH to the alkaline side, bacteria were usually found; similarly, Buhrmester declared that all her patients with pH 6.5 or below always had negative cultures. Both investigators expressed the belief that acidity as reflected in low values of pH is unfavorable to the growth of pathogenic bacteria, thereby furnishing evidence for the concept that nasal secretion in situ possesses a purposeful acid barrier against infection. Moreover, the work of Hansel demonstrates that in allergy it is especially significant that when the pH of nasal and sinal secretions falls low on the acid side, there is a complete disappearance of eosinophils, and when the pH returns to the alkaline side, there is a return of eosinophils. His investigation furnishes additional evidence that in recession from allergic attacks nasal secretion possesses a purposeful acid composition.

An important but imperfectly understood agent present in nasal secretions is lysozyme, a substance found in various body tissues and secretions that has the property of dissolving certain bacteria. Although lysozyme is most active against nonpathogenic organisms, it can attack pathogenic organisms when allowed to act in the full strength in which it occurs in some parts of the body. Fleming[11] demonstrated that lysozyme preparations not only dissolve susceptible organisms but kill them and inhibit their growth.

Action of lysozyme is influenced by hydrogen ion concentration, its activity being optimal at an acid pH. According to Hilding,[16] a sharp reduction of the lysozyme content of nasal mucus, with a consequent decrease in the antiseptic properties of nasal secretions, occurs during and preceding the invasive stages of the common cold (acute rhinitis). In some instances an increase in the lysozyme content of the secretion takes place on the same day as the beginning of subjective improvement. Since the action of lysozyme is known to be optimal at an acid pH, this may possibly explain why lysozyme is most active in the slightly acid nasal secretions of the clinically normal nose and why it is sharply reduced in the alkaline nasal secretions accompanying the acute common cold.

Hyperventilation is so common that its significance is frequently overlooked. Every person will at some time find himself in a state of hyperventilation.

Chemical reactions of the body are quickly altered by changes in the concentration of the carbon dioxide in alveolar air. Loss of the carbon dioxide of alveolar air produces alkalosis within the body. Alkalosis may be induced voluntarily by deep and excessively rapid respiration. Alkalosis occasionally occurs during hysterical attacks as a result of hyperventilation (blowing off of carbon dioxide).

Alkalosis due to hyperventilation has been observed in persons suffering from neurologic disorders, anxiety neuroses, and hysteria. Hyperventilation frequently accompanies rage, sorrow, fatigue, excitement, and apprehension. The effort of crying seems to be made easier by hyperventilation. Just as emotional changes can cause vasomotor changes in the nasal mucosa of some noses, so they can convert for short periods nasal pH from a normal, slightly acid state to an abnormal alkaline state.[4] Recently,[9] children were instructed to blow forty times against a sheet of tissue paper so that it would sway each time in response to the blowing off of carbon dioxide. Nasal pH readings were taken immediately before hyperventilation and immediately after the termination of the procedure. An alkaline nasal state ensued in many of the children and in most of those who cried.

Hyperventilation may also occur because of the increased respiratory rate associated with an increase in body temperature. It is prone to take place in inflammatory conditions involving the respiratory passages, especially in pneumonia, and presumably can explain in part the reason for the alkaline nasal pH occurring during acute rhinitis, acute sinusitis, and the more active stages of allergic rhinitis. It is during the time of acute nasal embarrassment that the nasal mucous membranes are inflamed or congested, the secretions are increased, and nasal breathing itself becomes more labored. Hence nasal alkalosis takes place in response to the act of hyperventilation (blowing off of carbon dioxide). The return to a normal nasal acid state ensues when hyperventilation no longer occurs and clinical nasal improvement follows.

pH FACTOR IN NASAL MEDICATION AND APPLIED CLINICAL PRACTICE

In recent years attempts have been made to direct the interest of rhinologists to the relation of the pH of nasal secretion in situ to applied nasal physiology, and to the importance of the pH factor in the field of nasal medication and applied clinical practice. Intranasal treatment by means of rational, physiologic nasal medication requires not only treatment of mucous membrane surfaces (including ciliary action) but treatment of nasal secretions (including the mucous blanket). Normal nasal secretion, including the mucous blanket, possesses a purposeful acid barrier against infection, for it has been observed that acidity as reflected in low pH values is unfavorable to the growth of pathogenic bacteria, and that nasal secretion possesses an acid composition when receding after allergic attacks.

Fabricant[5-7] conducted a lengthy series of experiments to determine the effect of a number of commonly employed nasal vasoconstrictors, topical anesthetics, silver preparations, and antiseptics on nasal pH. The pH of the nasal vasoconstrictors varied markedly from a highly acid pH 2.3 to an alkaline pH 9.5. The manner in which the trend of the nasal pH variation is affected depends on the pH of the drug employed. Thus, drugs with a highly alkaline pH induce in the nasal pH variation a pronounced drift toward alkalinity. Drugs with a pH range of 5.5 to 6.5 (as in the case of an appropriately buffered solution of 1 per cent ephedrine hydrochloride in physiologic sodium chloride) which ap-

proximate the normal range of nasal pH do not markedly affect nasal pH. Drugs with a pH level of less than 5 induce a drift downward in nasal pH, i.e., toward acidity.

Nasal vasoconstrictors possessing an unusually high alkaline pH value disturb the normal nasal pH level present in clinically normal nasal passages, enhance irritation of the nasal mucous membrane, and postpone the return of the nasal mucosa from a pathologic alkaline status to a normal, slightly acid status. During an attack of acute rhinitis, acute sinusitis, and the more active stages of allergic rhinitis, the employment of a nasal vasoconstrictor that lowers the abnormal alkaline pH in these conditions to a normal, slightly acid pH level between 5.5 and 6.5 in adults or 5.0 and 6.7 in infants and children—in short, a physiologic nasal pH—is most desirable in applied clinical practice.

SUMMARY AND CONCLUSIONS

Nasal vasoconstrictors occupy a commanding position among the various therapeutic measures employed by the general public for the relief of nasal congestion and the common cold. Although ensuing relief is temporary, overcoming the discomfiture caused by intranasal obstruction is welcome to distraught patients. There is little to indicate that the current passion for psychosomatic phrascology will alleviate millions of inflamed and allergic American noses. For physicians to disregard the opportunity of producing palliation, when such effect is easily possible, is tantamount to complete therapeutic neglect. Since the function of physicians is to prescribe measures of relief in the event a cure cannot be obtained, habitual rejection of the use of so simple a therapeutic device as a physiologic nasal vasoconstrictor in the treatment of intranasal congestion will often drive patients to over-the-counter purchases and self-medication.

In this investigation, the right nasal passages of forty rabbits received measured instillations of a progressively buffered solution of 1 per cent ephedrine hydrochloride in physiologic sodium chloride, the left nasal passages serving as a control, three times daily for a period of thirty days. The ephedrine preparation bore pH values, 2.5; 3.0; 3.5; 4.0; 4.5; 5.0; 5.5; 6.0; 6.5; 7.0; 7.5; 8.0; 8.5; 9.0; 9.5; 10.0; 10.5; 11.0; 11.5; and 12.0. Each of the animals was killed at the end of thirty days, the turbinal structures removed, and sections prepared for histologic study.

At autopsy, gross purulent exudate was demonstrated in animals treated with solutions of ephedrine buffered to pH 3.0, 3.5, 4.0, 4.5, 7.0, 11.5 and 12.0. In numerous instances the microscopic sections revealed a similarity between the ephedrine-treated and the untreated nasal mucous membranes, with histologic changes of a minimal character. For the most part, the pathologic changes in the respiratory nasal mucous membranes consisted of slight to considerable hyperemia; acute, subacute, and chronic inflammation; and varying degrees of suppuration. Although in this study neutral solutions of ephedrine produced inflammatory reactions, in general highly acid and highly alkaline solutions are more likely to produce traumatic and suppurative nasal mucosal changes.

In the human nasal cavity, the pH of nasal secretion in situ in clinically normal adult nasal passages ranges from approximately 5.5 to 6.5, whereas in infants and young children it ranges from 5.0 to 6.7. During acute rhinitis, acute sinusitis, and during the more active phases of allergic rhinitis, the pH of nasal secretion in situ is found on the

alkaline side and then shifts back to the acid state when the stage of clinical resolution is reached. It is during the time of acute nasal embarrassment that the nasal mucous membranes are inflamed or congested, the secretions are increased, and nasal breathing itself becomes more labored. Nasal alkalosis takes place in response to the act of hyperventilation (blowing off of carbon dioxide), and this presumably can explain in part the reason for the alkaline nasal state during acute rhinitis, acute sinusitis, and the more active stages of allergic rhinitis.

Intranasal treatment of the human nasal cavity by means of rational, physiologic nasal medication requires not only treatment of mucous membrane surfaces (including ciliary action) but treatment of nasal secretions (including the mucous blanket). Normal nasal secretion, including the mucous blanket, possesses a purposeful acid barrier against infection, for it has been observed that acidity as reflected in low pH values is unfavorable to the growth of pathogenic bacteria and that nasal secretion possesses an acid composition when receding after allergic attacks.

Nasal vasoconstrictors possessing an unusually high alkaline pH value disturb the normal nasal pH level present in clinically normal nasal passages, and postpone the return of the nasal mucosa from a pathologic alkaline status to a normal, slightly acid status. During an attack of acute rhinitis, acute sinusitis, and the more active stages of allergic rhinitis, the employment of a nasal vasoconstrictor that lowers the abnormal alkaline pH in these conditions to a normal, slightly acid pH level between 5.5 and 6.5 in adults or 5.0 and 6.7 in infants and young children—in short, a physiologic nasal pH—is most desirable in applied clinical practice.

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DISCUSSION

Leland G. Hunnicutt, M.D., Pasadera, Calif.: Dr. Fabricant has presented some well-worked out experiments on the use of a vasoconstrictor having different pH values. His presentation is timely because there is a tendency to swing too far away from the use of vasoconstrictors after having received so much information on the many evils that attend overuse and misuse.

His work follows the simple fact that living tissue maintains the best state if it does not have to contact irritants. The experimental work presents the effect of 1 per cent ephedrine of varying pH values on the mucous membrane of rabbits. His findings, in general, show greatest irritation from highly alkaline and highly acid solutions. There are a few unexplained reactions and lack of reactions. I found this to be true in using different types of vasoconstrictors in mice. Respiratory infections are common and give inflammatory changes identical with those of irritating vasoconstrictors or from their overuse. This effect from respiratory infection was brought to my attention through a personal communication from Kelemen. I made the mistake of having several mice in one cage and cross infection occurred more frequently. I feel that some of the changes result also from the vasoconstrictor effect. In some mucous membranes, this diminished blood supply by frequent and prolonged use of vasoconstrictors results in a noninfectious inflammatory reaction, giving the same appearance as that from infection or from too low or too high pH. I have found when using 0.25 per cent ephedrine in normal saline solution as nose drops in mice three to six times a day for ten days, there is usually little reaction but occasionally there is a violent inflammatory reaction. In studying a group of untreated mice, the incidence of inflammatory changes resulting from incidental respiratory infection is noted to be definitely less than when ephedrine is used. It is interesting to note that the inflammatory reaction is least with vasoconstrictors of short duration and greatest with those of long duration. This again bears out the statement that a noninfectious inflammatory reaction may occur in respiratory mucous membrane, probably due to an increase in catabolic products when prolonged ischemia prevents adequate circulation. This may be the answer to some of Dr. Fabricant's unexplained observations.

Dr. Fabricant has given a good summary of the more important research pertaining to mucous membranes. He has given good advice as to our role in observing and treating. Relief of certain symptoms may be obtained by judicious use of vasoconstrictors. His appeal to use proper pH must be followed if the patient is to get maximum relief and minimum irritation. I believe Dr. Fabricant has contributed greatly to our knowledge of treatment of mucous membranes. It has been a privilege to discuss his paper.

ALBINISM

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ALBINISM, with all its varying and labile expressivity, has long intrigued the laity as well as those of scientific discipline. The ocular aspects of the entity, aside from a few notable studies,[2,7,12] have actually failed to attract much research. It is the purpose of this paper to review the subject from the viewpoint of the ophthalmologist and to plead for a revival of scientific investigation. Much could be expected from a combined study utilizing the resources of the biochemist, the dermatologist, the geneticist and the ophthalmologist, for surely our knowledge is scant.

Albinism is the clinical expression of a gene-determined inability of the individual to convert precursor substances into melanin pigments, a so-called inborn error of metabolism.[5] While authorities are not in complete agreement, it appears that melanin metabolism develops in a step-wise fashion[9] with compounds being converted into other compounds. These compounds, or specific units in the chain of chemical transformation of protein into melanin, are under the control of specific genes. Mutation of a gene or genes controlling such unit processes may lead to blocks at various points and result in an inability to produce certain normal compounds. There may result an abnormal accumulation of precursor substances, normally converted into other products of degradation in this series, which exhibit clinically detectable evidence suggesting the metabolic dyscrasia. Alkaptonuria, tyrosinuria and phenylketonuria are such known errors of inborn metabolism. Many workers, especially those interested in chemical genetics, support the belief of a one to one relationship of a gene to each specific step in the metabolic reaction chain. In the literature it is constantly repeated that albinism is the sequel of an absence of the enzyme *dopa-oxidase*, an inability of the individual to further convert dopa to melanin. Lerner and Fitzpatrick,[6] in a recent summary of the subject of melanin production, have suggested that tyrosinase is the only enzyme responsible for the conversion of phenylalanine and tyrosine into melanin and that albinism is specifically the result of the absence of the enzyme tyrosinase. They insist that the existence of the enzyme *dopa-oxidase* has not been demonstrated. A closer scrutiny of the process of conversion of the protein substrate to melanin discloses that it is extremely complicated. The presence of the following known substances is actually required: (1) the enzyme tyrosinase, (2) copper for the activity of the enzyme and (3) a suitable and adequate substrate, presumably tyrosine. To further complicate the picture the enzymatic reaction is controlled by several physicochemical factors which include (1) oxidation-reduction potentials and (2) naturally occurring sulfhydryl compounds in the epidermis. Many of these accessory factors in the formation of melanin may be assumed to be gene-controlled, aberration of which could induce variation in the end

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product and possibly account for the many clinical varieties of human albinism.

It is entirely conceivable, for example, that albinism could result if abnormal sulfhydryl compounds existed in sufficient quantity to inhibit the action of the enzyme tyrosinase, even though the latter was present in normal concentration.

CLINICAL TYPES OF ALBINISM

It is almost impossible to fix a specific limit at which albinism begins and normal pigmentation ends. Human albinism exhibits a wide range of grades of pigmentation in the eye, hair and skin. It has been suggested that "it is actually difficult to regard albinism as a general absence of an enzyme but rather to regard the process as a complete or even local structural or physiological differentiation which prevents the possibility of some metabolic process."[7]

Many classifications of albinism are available for study. Sorsby's conception[8] with some modification is herewith presented for your study.

A. Generalized Albinism

1. Complete Generalized Albinism
   a. Total or nearly total lack of pigment throughout the entire body
      Heredity
      (1) Recessive-autosomal
      (2) ? intermediate (incomplete recessiveness)

2. Incomplete Generalized Albinism
   a. The ability to produce a minute amount of melanin is present and this pigmentation accumulates with advancing age
      Heredity
      (1) Recessive-autosomal
      (2) Irregular dominant
      (3) ? intermediate (incomplete recessiveness)

3. Albinoidism—Leucism
   a. Very light hair and skin, no nystagmus or photophobia, good vision, blue irides
      (1) Increasing pigmentation of hair, skin and eyes with advancing age
      Heredity
      (1) Autosomal dominant

B. Albinism Confined to Globe of the Eye

1. Entire Eye Albinotic
   a. Head nodding, nystagmus, usually defective vision, pink to blue irides, iris translucency, normal pigmentation of the skin and hair
      Heredity
      (1) Sex-linked recessive

2. Albinotic Fundus Only
   a. Head nodding, nystagmus, usually defective vision, blue to brown irides, some iris translucency
      Heredity
      (1) Sex-linked recessive
      (2) Intermediate sex-linked

GENETICS OF ALBINISM

In man, albinism, as in so many other of his genetic aberrations, exhibits an interesting interchangeability of hereditary patterns.

Complete generalized and incomplete generalized albinism are most frequently transmitted by an autosomal recessive gene. A perusal of the literature permits one to speculate that several different genes have mutated to produce this type of albinism (different genotypes).

The incidence of albinism varies from one in 5,000 to one in 25,000 and is world wide in its geographic distribution. The rare occurrence of albino marrying albino and yet producing normally pigmented children[10] would suggest, as one explanation, unlike recessive genes. There is some evidence that a dominant autosomal gene exhibiting irregular penetrance may be responsible for incomplete generalized albinism in some families.

The possibility that the gene in its heterozygous state (the carrier parents) may exhibit some clinical evidence of its presence has been suggested by Waardenburg.[13] The author demonstrated that certain heterozygous individuals (both for the generalized and ocular albinism) readily transmitted light through the irides upon transillumination of the globe. This incomplete expression of the gene's presence would imply an intermediate form of transmission.

Albinoidism presents a dominant autosomal type of inheritance. The hair, skin and eyes simulate closely that of the generalized albino, but usually the vision is good and the iris is seldom translucent. Nystagmus, or photophobia, is seldom present, but may be so.

In albinism confined to the globe the hereditary transmission appears to be x-chromosomal (sex-linked). Waardenburg has written that he can also demonstrate iris translucency in the heterozygote (or carrier female) of this type of albinism which again suggests an intermediate inheritance pattern. In Falls' experience[4] with ocular albinism, chiefly confined to the fundus, only the female heterozygote actually presented a clinically recognizable change in the fundus (fig. 1). This was characterized by the presence of small islets of coarse, cocoa-brown pigment clusters, separated by considerable expanses of visible choroidal vasculature. The macular areas

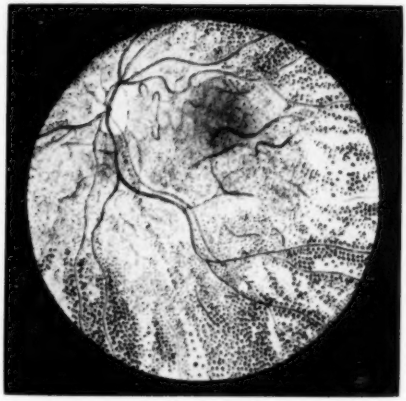

FIGURE 1.

were stippled, indicating lessened pigment formation, and the foveal reflex was easily visualized. The vision in these carrier females was normal. Thus again an intermediate inheritance pattern is suggested.

OCULAR MANIFESTATIONS OF ALBINISM

Clinically the albino eye is characterized by a pink, translucent iris, red pupils, nystagmus, photophobia, defective vision and usually a considerable degree of error of refraction. These factors may exist in all combinations and degrees of severity. Moreover, with advancing age, amelioration of the signs and symptoms may be encountered apparently hand in hand with increasing pigmentation. The hair is usually white, or yellowish white (rarely reddish) and the skin pinkish white. The eyebrows and eyelashes are usually white with an associated pinkish conjunctiva. A rather wide range of variability exists between individuals of unrelated families, but considerable similarity exists between affected members of related families.

Iris and Pupil

The pink pupil and iris are largely a consequence of reflected light from the

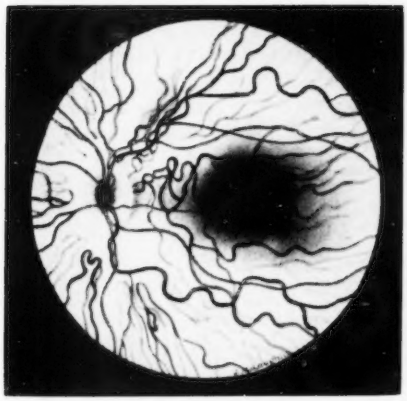

FIGURE 2.

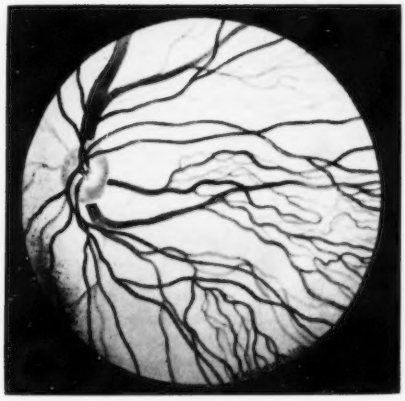

FIGURE 3.

choroidal vasculature. Red, white, or greyish admixtures exist depending upon the thickness of the iris and the extent of pupillary dilatation. It is rare, indeed, to observe complete lack of pigmentation of the neuro-ectodermal iris leaf when the iris is subjected to careful biomicroscopy. The position of the light, thickness and texture of the iris all influence the color.

Fundus

The fundus likewise exhibits a great deal of variability of appearance, usually presenting a bright orange-red color, with marked prominence and vivid visibility of the choroidal and retinal vasculature. In the great majority of cases the macular area exhibits a more uniform pinkness with slight masking of the underlying choroidal vessels (figs. 2 and 3). The optic nerve is usually reddish in appearance and seems slightly darker as a result of contrast with the surrounding retina. In our experience the greater the macular pinkness and masking of the choroidal vessels the more efficient are the visual acuity and ocular function. There is some evidence that this macular pigment (clinical presumption) increases with advancing age, especially in incomplete generalized albinism and albinism of the eye alone.

Visual Acuity

Visual defect is almost always very marked in the generalized albino eye while frequently it is less seriously defective in the incomplete generalized albino. In the sex-linked ocular types of albinism a wide range of variability exists, but usually the acuity is nearly as defective as in the generalized albino. Individual exceptions are always to be expected. In one of our sex-linked ocular albino families an affected male individual exhibited 20/30 + O.U. visual acuity and his fundus closely simulated the female heterozygote appearance.

It has been clinically noted by us that even very blond children will frequently exhibit improvement in visual acuity with the accumulation of pigmentation during the early childhood years. Amblyopia in the range of 6/9 to 6/12 may occasionally be attributed to incomplete development of the macular area subsequent to defective or lessened retinal pigmentation at birth.

Most authorities point out that the marked dazzling which the albino eye is

subjected to as a consequence of the absence of pigment protection is partially responsible for the poor visual acuity. The author, however, has not been able to improve to any measurable degree the acuity of albinos with the use of shields, colored contact lens and so forth, despite reports in the literature to the contrary. Telescopic lenses, on the contrary, have in our experience been a most valuable adjunct to these patients.

The great number of albinos will be, or are, able to read fairly satisfactory type (Jaeger 1 to 4) despite their amblyopia for distance. The ophthalmologist must, therefore, exert marked caution in recommending such children for blind school education and Braille. There are in our files the records of three albinos who have secured their master's degrees, having attended regular public schools and universities. It must be admitted, in all fairness, that an occasional albino will need special education to prepare him for a vocation.

Errors of Refraction

It is most interesting to note that by far the greater proportion of albinos exhibit a considerable degree of refractive error, both hyperopic and myopic. Astigmatism of a high degree is commonly associated. The more complete the albinism is on the whole the greater the degree of refractive error.

Photophobia

This feature of the albino eye seems to be a consequence of "dazzling introduced by the lack of contrast between the retinal impulse and the surrounding media."[7] As has been previously observed, there exists only minimal intrafamilial variability of this symptom in contrast to the greater variation between unrelated individuals. The generalized albino usually exhibits the greatest degree of photophobia.

Visual Fields

Visual fields in albinos have usually revealed a central scotoma of varying degrees of density. No comparative studies of the visual fields in various types of albinism could be found. Edmunds[3] found that the form fields of his albino group exhibited temporal contraction.

Color Vision

Color vision studies in a pooled series of albinos by several authors have suggested that the albino eye presents no greater incidence of color vision defect than does the normal eye. Edmunds' study[3] indicated that the poor visual acuity of the albino often results in low color discrimination.

Dark Adaptation

Dark adaptation studies performed on albino subjects have not revealed any specific abnormality in this phase of visual physiology.[1,3]

Nystagmus

Nystagmus is an almost constant feature of the generalized albino eye. The nystagmus is usually horizontal, with or without a rotatory component. A wide range of amplitude and frequency is encountered both in the same individual and between affected individuals. Clinical experience, both personal and in the literature, is that nystagmus becomes less severe with advancing age and with increasing pigmentation.

Strabismus

Strabismus is not an uncommon feature of the albino, both divergent and convergent forms being encountered. The literature suggests that the incidence of divergent strabismus is slightly greater. The lack of macular tone in the albino could well account for both the high incidence of nystagmus and strabismus encountered.

Microscopic Study

Careful microscopic studies of the albino eye have not revealed any specific anatomic change incompatible with the normal eye other than the lack of pigmentation. A few observers have reported a hypoplasia or aplasia of the macular area. This latter finding lends support to Vogt's hypothesis[11] that the albino eye lacked the yellow macular pigment when examined with red-free light.

Each of the previously mentioned signs and symptoms of the albino eye may exist alone or in varying combinations occurring either sporadically or in several members of a family. Albinoid fundi are quite frequently observed linked with nystagmus and high errors of refraction (myopia, hyperopia and astigmatism). Hereditary transmission of these combinations is to be anticipated.

DISCUSSION

It must be apparent to the discriminating physician that the heretofore adhered to dictum that all albinism is recessively inherited must be discarded. That a rather universally distributed recessive gene for albinism does exist is indicated by the appearance of albino offspring from intermarriages between the red, black, brown, yellow and white races, and most complete generalized albinism is the result of this specific gene. The appearance of pigmented offspring, however, from the marriage of apparently similar generalized albinos and the reported high consanguinity rate (20 to 33 per cent) suggests that several different recessive genes actually exist for the trait. In addition, dominant pedigrees for incomplete generalized albinism have been reported. Albinism confined to the eye alone is sex-linked (x-chromosomal) recessive as well as intermediate in its pattern of transmission. The affected individual exhibiting albinism of the eye (alone) may not infrequently exhibit incomplete albinism of the hair and skin, especially during infancy and early childhood. No ophthalmologist, consequently, should undertake to give eugenic or even prognostic advice to these individuals or families until he has (1) a carefully taken pedigree of the family, (2) meticulously examined the affected patient and (3) examined at least the patient's siblings and parents.

It is our limited experience that the ocular signs and symptoms of the albino vary, in lessening degree of severity, with advancing age in the following order in respect to the clinicogenetic type of albinism:

(1) Complete generalized albinism (recessive - intermediate)	(2) Incomplete generalized albinism (recessive - intermediate-dominant)
(3) Albinism confined to the eye (sex-linked recessive and intermediate)	(4) Albinoidism (dominant)

Most albinos seem to accumulate some melanin with advancing age. An amelioration of their signs and symptoms appears to go hand in hand with this pigment accumulation. Even visual acuity may improve to a limited or moderate degree. This latter change may perhaps explain the etiologic basis for those cases of 6/12 to 6/9 visual acuity who exhibit normal fields and fundi. The history of several of these patients has indicated that nystagmus and marked blondness existed in early childhood.

The ophthalmologist should always keep in mind the association of nystagmus, head nodding, photophobia, and high errors of refraction with albinism. Patients presenting any of these symptoms should be scrutinized most care-

fully for evidence of generalized or ocular albinsim. Waardenburg's demonstration of diaphanous irides in the carriers (heterozygous individuals of albinism) compels the oculist to utilize the simple procedure of scleral transillumination when such is suspected.

Therapeutic management of the albino eye has to date been chiefly palliative: tinted glasses, colored contact lens, tattooing of the sclera and cornea, inserting of pigmented foils in the eyelids and so forth. In a large majority of the albino eyes encountered by us the visual function can be measurably improved with the prescribing of telescopic lens. Recently hormonal and enzymatic therapy has permitted the albino to tolerate sunlight more comfortably and has effected a reduction in his photophobia. The chief and most troublesome complication is liver damage, but it is to be anticipated that with added experience and research we may yet treat these albino patients early enough in life (first three months) to secure sufficient macular development with subsequent useful visual acuity.

SUMMARY

The ocular aspects of albinism are reviewed. It has been suggested that the clinical picture (phenotype) exhibits a rather wide range of variability. This is further substantiated by the appearance of many different hereditary patterns (genotypes). Sorsby's classification has been presented in modification. The oculist is advised to anticipate albinism as a possible etiologic agent in photophobia, nystagmus, high errors of refraction, head nodding and reduced visual acuity.

It is suggested that most of the signs and symptoms of albinism improve with advancing age and with the accumulation of pigmentation.

Encouraging therapeutic advances in albinism seem probable in the not too distant future.

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DISCUSSION

JOHN CLEMENT MCCULLOCH, M.D., Toronto, Ont., Canada: Dr. Falls has given a lucid description of a group of diseases which frequently are taken for granted and, therefore, are little studied. Complete albinism is so rare and so striking that the ophthalmologist can easily slip into the habit of classing patients as albinos or normals and inquiring no further. Partial albinism is commoner and more interesting than complete albinism. A visual acuity of 20/40, not correctable, may occasionally be explained by albinism confined to the eyes. A patient with moderate albinism may have fairly good visual acuity and, if he is young, his acuity will improve as he grows older. In the normal growth of a child the transition of infant's vision to the adult level of acuity possibly awaits the completed expression of pigment in the fundus. Indeed, a closer inspection of the subject quickly leads to further knowledge and further speculation.

The problem can be illustrated by the difficulty of classification. Albinism could be classified on an anatomic and clinical basis, on a chemical basis, or by the hereditary variations. At the moment, an anatomic classification is possible and is used by Dr. Falls. The weakness in this organization lies in the fact that there is not a clear-cut separation between the groups. The point where complete albinism ends and partial generalized albinism starts, and, in turn, where localized albinism can be distinguished from generalized albinism, is not easy to decide. Added to this difficulty is the fact that with intermediate inheritance the signs in the homozygous affected person are more severe than in the heterozygous patient.

Classification on a chemical basis is not yet possible. However, as knowledge accumulates assessment of cases from this point of view would be more basic than from the anatomic approach. There are several steps in the chemical sequence leading to the formation of pigment. A classification of albinism according to the defect in this chain of reactions would be directed to the very foundation of the trouble.

While classification on an anatomic basis is unsatisfactory, and on a chemical basis is not yet available, organization on a genetic basis is fundamental and is rapidly becoming possible. Dr. Falls has already indicated the dominant, intermediate, recessive, and sex-linked types. Studies of cross inheritance will further clarify the situation by pointing to the autosomes which are affected. As knowledge of the inheritance of albinism increases, and as the details of the defects in chemistry are filled in, the relation between those two will become clearer, and the various clinical pictures will be found to reflect the basic deficiencies.

DR. FALLS: I wish to thank Dr. McCulloch for his very excellent additions to this paper.

I want once more to emphasize that I think every albino child should be most carefully scrutinized from the ophthalmologic point of view before he is confined to a school for the blind. It is a very unhappy experience to find albinos who can read readily and without difficulty subjected to a blind school education.

HEERFORDT'S SYNDROME

GEORGIANA D. THEOBALD, M.D.
OAK PARK, ILL.
and
HOWARD L. WILDER, M.D.
CHICAGO, ILL.
BY INVITATION

MRS. H. K., age 54, had good vision until 1948 when she developed bilateral uveitis accompanied by parotitis, facial and partial throat paralysis.

Her first symptoms were migratory arthritis and fever. These were followed by multiple skin lesions, bilateral uveitis, parotitis and the facial and throat paralysis. In one year's time, she had no useful vision in either eye, and three operations had been done for the relief of tension.

She was first seen at the Illinois Eye and Ear Infirmary of the University of Illinois in January 1950. The vision of each eye was light perception and poor projection. There was mild conjunctival injection. The cornea was diffusely cloudy and vascularized. The sclera was white. There were many fluffy corneal percipitates. The iris was thick and bound down to the lens. The fundus could not be seen. The diagnosis of Heerfordt's syndrome was made. An optical iridectomy was made on the right eye with slight improvement of vision.

The patient's family history included the information that one sibling died of multiple sclerosis, three siblings died of tuberculosis and her husband had a lung abscess.

In March 1950, she was admitted to the Research Hospital of the University of Illinois almost comatose due to renal insufficiency. All tests for tuberculosis were negative. Also tests for brucella, typhoid and tularemia were negative. A skin biopsy of a lesion of the right thigh was consistent with Boeck's sarcoid.

Films of the right foot showed several small defects in the bone at the distal and proximal phalanx of the first toe which might have been related to Boeck's sarcoid. Roentgenograms of the chest showed a number of small densities throughout both fields. Skull, sinus, and dental films showed nothing abnormal. A diagnosis of generalized sarcoidosis was made. Her response to treatment was good.

In December 1950, she was readmitted to the Illinois Eye and Ear Infirmary for enucleation of the left eye, on account of a spontaneous rupture of an anterior staphyloma. The right eye at that time showed light perception and projection and band-form keratopathy; tension was hard; 2 + beam in anterior chamber; the fundus was not seen.

A three months' course of urethane was followed by marked systemic improvement. Roentgenogram of the chest in February 1952 showed nothing of significance.

She was last seen in June 1952 and found to be limited physically only by the ocular condition.

Presented as a *Clinicopathologic Case Report* at the Fifty-Seventh Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 12-17, 1952, Chicago, Ill.

MICROSCOPIC FINDINGS

The circumcorneal area is thickened by fibrosis, hyperemia and infiltration of chronic inflammatory cells. On one side is a mass of epithelioid and giant cells extending to the rectus muscle.

The corneal epithelium is irregular and keratinized. There is a peripheral pannus. The stroma shows interlamellar infiltration and vascularization. The lamellae are atrophic and on one side the cornea is almost entirely replaced by scar tissue. Bowman's and Descemet's membranes are in folds and the endothelium is absent.

The anterior chamber is shallow centrally and the angles are obliterated by root synechias. A hyalinized connective tissue membrane which is closely adherent to the posterior cornea fills a good portion of the anterior chamber. This membrane contains granulomatous exudate with many giant cells.

The iris is atrophic and is replaced and infiltrated by a granulomatous mass containing a large number of plasma cells, lymphocytes, and epithelioid cells with many multinucleated giant cells. There are also some plasmocytoid cells and Russell bodies. There is no caseation.

The ciliary body is widely infiltrated with granulomatous tissue as described above, particularly prominent on the surface of the hyalinized processes, from whence it is continuous with that seen in the iris and anterior chamber. The infiltration is marked by the presence of many Langhans-type giant cells along the ciliary epithelium and the pars plana. There is senile proliferation of the nonpigmented epithelium.

The choroid is hyperemic and edematous, there is minimal infiltration with chronic inflammatory cells and the choroid is detached by hemorrhage and serous transudate.

The retina is detached posteriorly in the region of the disc by a hemorrhagic exudate. There are marked atrophic changes with vacuolation of the inner and outer nuclear layers. There are several large hemorrhages in the nerve fiber layer.

In the disc, the lamina is retroplaced and the deep glaucomatous cup is filled in with glial proliferation. The nerve shows disorganization of the glial columns.

DIAGNOSIS

1. Granulomatous noncaseating iridocyclitis and episcleritis.
2. Healed perforation of the cornea with intercalary staphyloma.
3. Detachment of retina and choroid.
4. Vitreous hemorrhages.
5. Glaucomatous atrophy of optic nerve.

The above granulomatous findings are consistent with Boeck's sarcoid.

The clinical picture of parotitis, facial palsy and granulomatous iridocyclitis was described by Heerfordt.

HOW TO DEVELOP A HEARING CONSERVATION PROGRAM

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BY INVITATION

WE REALIZE that opportunities for developing a hearing conservation program in a large sparsely populated area like Arizona with a heavy Indian and Mexican population may not be comparable to opportunitites in some metropolitan areas. On the other hand, we think that the basic principles of organization are the same for all areas, and in the following discussion of the development of the hearing conservation program in Arizona something may be learned from the mistakes we made and from our experience in the program that was developed.

The hearing conservation program in Arizona is the result of a lot of hard work and planning by many different individuals and groups. It required teamwork, and never could have been accomplished without the help and active participation of several hundred people. It is impossible to give you all of the details of organization in such an extensive program in one short paper, so we have prepared a syllabus on "How to Develop a Hearing Conservation Program." It includes some of the materials that were used in the Arizona program. Some of these materials are original, others have been so effectively used in other programs that we adopted them for our use.

In reading the outline one is impressed by material that appears extremely simple, for it contains facts that everyone should know. But one of the things that we learned early was that we could not take too much for granted in any of the areas of planning. For example, in the first year of our program the hearing director assumed that the school nurse knew when to send out letters telling about the medical examinations, and in preparation for writing the letters and planning the follow-up an intensive training period with the nurse was carried out. The director of the program felt confident in leaving town for a few days to attend a meeting on the West Coast, leaving the responsibility of sending the letters to the nurse. However, when she returned the entire town was up in arms, wanting to know what was going on. The otologist was angry and asked "What's going on here? These organizations are always stirring up things. These children don't have a hearing loss." Upon investigation the director learned the reason for the furor. The nurse had sent the wrong letters, sending the ones that notified the parents of the otological examination following the screening test, rather than following the final threshold test. Naturally the otologist was upset. Children, some of whom were his regular patients, appeared in his office in groups with their parents, and indeed, some of them had no hearing loss.

The director went to see the otologist immediately to explain what had happened. Together the director and otologist checked the standards of referral for otological examination in the syllabus used as a guide in developing the program.* When the otologist learned that professional standards, procedures

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and technics were being used, he was glad to examine the children and to act as medical advisor for the program in that area. The director and the otologist then worked out a plan to inform the public of the error. First, the director assumed all of the blame and called on the parents of each child involved to explain the situation. The newspaper cooperated by publishing a front-page story explaining the error in the letters and describing each step of the hearing conservation program in detail. This comprehensive story probably would not have been achieved without the mistake, and it cost only an afternoon of the consultant's time to test the hearing of the newspaper staff, including the editor and the owner-manager. However, this incident exemplifies one of the times during the development of the five-year program when it was discovered that a simple detail overlooked or one mistake in any area of the planning can cause immeasurable trouble.

In starting a hearing conservation program such as the one developed in Arizona, where does one begin? First, there must be an interested group to serve as sponsor and to employ a qualified director. This may be a group of otologists, a public health service, the American Hearing Society, an endowed foundation or a private society. In Arizona the sponsor group was the Arizona Society for Crippled Children and Adults, Inc. There were no services for the acoustically handicapped child in the State Health Department, in the Department of Public Instruction or in any other agency, public or private. The State Health Department requested the Arizona Society for Crippled Children to develop such a program, and the second year the State Medical Association joined the Society in sponsoring the program. The Arizona Society for Crippled Children employed the director, who conducted the testing program and the educational follow-up. The Hearing Conservation Committee of the State Medical Association planned the otological examinations and treatments. The original request from the Health Department was for a survey in Arizona of children with hearing losses. However, the Society for Crippled Children and the State Medical Association were in agreement that just a survey to discover the children with a hearing loss had little real value. They were also concerned with a sound program for the children, including medical and educational follow-up. One of the major problems in Arizona was how to reach all of the children in an area larger that Indiana and Michigan combined, the population of which is less than that of Seattle, Washington, or Providence, Rhode Island, and less than one third of the population of Detroit, Michigan. Among the methods studied for reaching all the children were mobile clinics. However, they proved too costly for the funds available, and the follow-up possible in the vast rural area was also questionable. There was no trained personnel in the State for testing and almost no equipment. The State Health Department had three used 4-C Western Electric group speech audiometers and one very old pure tone audiometer. The Arizona Society for Crippled Children had the equipment checked and calibrated and the testing began in October 1946.

Before the testing program began, all key people who were involved in the program were contacted and the program thoroughly explained to them. They helped with the plans. Being primarily a hearing conservation program

* "Syllabus of Audiometric Procedures in the Administration of a Program for the Conservation of Hearing of School Children" edited by Horace Newhart, M.D. and Scott N. Reger, Ph.D., for the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology, April, 1945.

for school children, the plans were made by the hearing director with the cooperation of the State Superintendent of Public Instruction. The Superintendent of Public Instruction sent out notices to local superintendents, supervisors, principals, and head teachers, telling them about the program, its purpose, aims and sponsors. He suggested that the procedures and details be worked out at the local level with the hearing director. Workshops for teachers were planned and were often arranged for through the State Superintendent of Schools. These workshops were found to be invaluable in the educational follow-up where educational problems of the hard of hearing child were studied.* Officials in the Public Health Department were consulted. The program planning that involved public health nurses was worked out with the Supervisor of Public Health Nurses in the state. Plans for the care of indigent children with a hearing loss were developed with the heads of the Department of Public Welfare. All plans and arrangements were made at the state level and were handled through the county welfare units at the local level. In the second year of the program the otological examinations and treatments were handled by the otological committee of the State Medical Association through the proper channels of the county medical societies. The State Director was invited to assist with the planning of the medical program in order to insure continuity of organization.

As the program developed, we learned that it was important to keep all departments involved in any way in the program informed of the procedures, and to work through the proper channels to the local level. This insures thorough understanding and cooperation at each level. When department heads change, the new ones should be contacted and oriented immediately. Such changes occurred often in Arizona. There were four Superintendents of Public Instruction between 1946 and 1950, two Superintendents of Public Health, and every year a new President was elected in the State Medical Association and in each County Medical Unit.

The first year of the survey, 14,905 children were tested in four counties with the Group Speech Audiometer; 1,352 were given pure tone follow-up tests and 543 were referred for otological examination. The group-testing method required one-third or more retests in each group, and considerable time was necessary to plan and carry out the testing. Earphones were often unreliable. Scratched records and noise in the testing apparatus were hazards to reliability. At the beginning of the second year the group equipment became even more of a nuisance. It required too much time to keep it in repair. Rechecks were made three or four times in an effort to ensure reliable results. In the middle of the second year when an intensive program was started in a concentrated area, the Tucson Public Schools, it was decided to try all testing, including screening, with the individual pure tone audiometer. This method required the services of a larger number of trained audiometrists. A series of courses in pure tone audiometry was set up for the school and public health nurses in the Tucson area. The nurses screened the entire school population, which consisted of approximately 20,000 children, with individual pure tone instruments in less time than they had previously taken to screen the same number of children with the group speech phonograph machine, when they had screened forty children at a time.

* See syllabus "How to Develop a Hearing Conservation Program," Section on "Suggestions to Parents of Children with a Loss of Hearing."

In 1947-1948, the second year of the survey, a total of 22,475 children were tested, the largest number tested during any one year of the program. Of that number, 663 tests indicated a hearing loss. The criterion used for referral for medical examination was a 20 decibel hearing loss in two tones in the speech range, or a 30 decibel loss in one tone in the speech range. Thirty decibel losses or more, beyond 4,000 cycles, were referred for medical examination. If no pathologic condition or treatment was indicated, the loss was recorded and the hearing rechecked every six months. If the loss remained constant for two years, the child was not referred for future medical examination. Individual pure tone screening required more trained personnel and equipment and, hence, more money. During this time an extensive public education program was in progress. Demonstrations and talks at service clubs, Rotary, Lions and other clubs, resulted in the purchase of more pure tone audiometers. Press and radio programs helped to inform the public of the needs in the field. Lectures and demonstrations at PTA meetings, women's clubs, church and other community groups served to make parents "hearing" conscious, and to recognize the need for early medical care when a child has a sore throat or earache. A characteristic remark of a parent in one of these meetings was, "I thought all children had earaches and sore throats as a part of growing up. I just put some cotton in the ear, maybe a little oil, and let it go at that. I never realized the danger I was courting." This mother, like hundreds of others, was conscientious and sincere, but uninformed. How can we expect parents to take their children to doctors for treatment or do anything about prevention of serious difficulties if they are not aware of the need? We cannot emphasize too strongly the value of a scientific and well-planned public education program in our efforts to conserve the hearing of children.

In the schools where the duties of the school nurse include everything from first aid to truant officer, it was not possible for them to devote as much time to any one program as the Tucson nurses devoted to hearing conservation the first year. Every child in school was tested and followed up. The need for more trained personnel, especially for routine testing, became evident. It seemed at first impossible to recruit enough personnel. There were no more nurses to train. In an effort to meet this need, it was decided to consider volunteer help for screening tests.

We entered this new venture with caution, as we were aware of the objections to the use of volunteers. We were told by professional people—doctors, nurses and teachers—that "volunteers get in the way, are irresponsible, unreliable and in general do more harm than good." But the pioneers who were concerned first of all with helping the children decided it was worth an experiment, and a volunteer training program was set up. A high standard of selection of volunteers was used. Most of them were college graduates, and some were former nurses and teachers. Some were doctors' wives. In one volunteer training course there was a retired doctor. She was still young, however, living in Arizona for health reasons. These women were intelligent, alert to the needs in the community, and willing to do something about them. They were not social climbers but were people who had leisure time and wanted to use it constructively.

We learned many things in this training program. One important thing was that volunteers wisely and carefully selected, well-trained and supervised, are invaluable to a program. We also learned that volunteers carelessly select-

ed, ill-trained and not supervised are a liability and may harm a program.

Another important thing we learned was that just anybody cannot test the hearing of children effectively and, furthermore, that nurses' training or other specialized training is no criterion. For example, in one of the large isolated communities the Kiwanis Club was helping to support the program financially. They were anxious to have a well-trained audiometrist do the testing in their schools and they thought a nurse would be ideal. They arranged for her to take the courses and were paying her to do the job. This nurse was an intelligent, fine individual, but she did not have an aptitude for giving hearing tests. She was all thumbs at the panel of the audiometer. She finally gave up voluntarily, saying, "I believe in the work, I will do anything I can to help the program, but I can't run that audiometer." Fortunately, this person recognized her lack of aptitude for this specific job and resigned.

Hand-picking individuals who had an aptitude as well as the other necessary requirements for a good audiometrist from a select group of people was a distinct advantage. A volunteer understood at the outset that if she did not have all of the required abilities necessary to give hearing tests, she would be transferred to another committee where her abilities could be used more effectively. As a result, the program was strengthened and extended to areas that could never have been reached without the assistance of volunteers.*

The in-service training courses in audiometry were so popular they became an integral part of the program. Nurses from the Public Health Department, the schools, and nurses from doctors' offices enrolled for them. In two and a half years every school nurse and public health nurse in the state had taken at least 12 hours of training, which included practice in testing the hearing of school children under supervision. Judging by the popularity of the courses among the nurses it seemed advisable to include testing the hearing of school children and other phases of audiometry as a part of the basic undergraduate training program for nurses. Such a program was presented to the Supervisors of Nurses' Training at the larger hosital in Tucson and Phoenix. It was adopted and proved a beneficial move, not only for the student nurses but for the hearing conservation program as well. The nurses assisted with the testing program in the schools during their period of supervised testing. By the time each nurse had spent four days testing in the field she had screened between 800 and 1,200 children. Some of the nurses made a special request for advanced courses in audiometry in order that they might learn to give the follow-up threshold tests.

The courses were organized on the following plan: First hour, general information and history of hearing conservation, the needs, the program in Arizona, the sponsors, purpose, etc. Second hour, demonstration and study of the audiometer. Third hour, technics in giving the hearing test. This section included some discussion on understanding the child and technics in working with children in the testing situation. Fourth hour, practice on machines, testing of each other. Fifth hour, demonstration testing with children. Sixth hour, demonstration of materials and discussion of technics. Second day, testing under supervision, six hours. This course was limited to ten students. The advanced course was offered to individuals who showed particular enthusiasm and aptitude in giving hearing tests, after the individual had experience in

* See syllabus "How to Develop a Hearing Conservation Program," Section on "Volunteer Training Program for Testing the Hearing of School Children."

screening approximately 500 children. The advanced course included 12 hours of theory, demonstration and practice in giving threshold tests. First hour, purpose and importance of audiogram, how it is used, and study of audiogram, technical aspects of construction of chart, and recording. Second hour, technics of giving the threshold test. Third hour, bone conduction test. Fourth hour, demonstration in charting audiograms. Fifth and sixth hours, testing and making threshold tests, charting and making audiograms under supervision. Second and third day, testing, making threshold tests under supervision. The advanced course was limited to only five students.*

During the testing period, under supervision, students were rated on (1) aptitude in handling the audiometer, (2) efficiency in giving the hearing test, (3) accuracy in the result and success in getting the response from the child, and (4) rapport with the child and other school people. Failure to pass on any one of these points would disqualify the individual.

By the middle of the second year the program was still without medical organization on a state-wide plan. The State Health Department officers made an attempt to work out a plan with the local county medical societies without success. The hearing director also met with them. The doctors sat with stone faces, sometimes grunting now and then, but they obviously were not interested in discussing any medical plan within a group to help hard of hearing children. They feared socialized medicine and would do nothing about working out a plan together. The hearing director proceeded to work with the doctors individually in each community, explaining the testing program in detail and soliciting their help for the medical examinations and treatment. The director soon discovered that the doctors were just as interested in the children in their own community as were the parents and the school people, and in every case the otologist examined the children without charge and worked out a plan for medical treatment with the parents. One otologist in the northern part of the state examined the children in that area, and one examined the children in the southern part, each working out the plans for medical treatment with the parents in their respective areas. The children in the adjoining counties where there was no resident otologist were transported by the local lay hearing conservation committee of the Arizona Society for Crippled Children to one of the abovementioned otologists for examination.

By this time the hearing conservation program was functioning in six out of fourteen counties. However, as the time came for the program to be developed in the more thickly populated counties, a definite medical plan of organization was necessary. A request was then made by the State Health Department and the Arizona Society for Crippled Children to the Professional Committee of the State Medical Association to appoint a State Hearing Conservation Committee in the State Medical Association to set the policies and plan the medical treatments.

In summary, the characteristics of the development of a hearing conservation program in Arizona and perhaps the unique differences from other hearing conservation programs are (1) the sponsorship by two private societies: the State Medical Association and the Arizona Society for Crippled Children; (2) an intensive, state-wide training program for audiometrists, parents and teachers on "Hearing loss in children"; (3) a strong state-wide continuous public education program and (4) the use of volunteers for testing.

* See section in syllabus on "Introductory Course on Testing the Hearing of School Children."

HOW TO ORGANIZE YOUR STATE MEDICAL ASSOCIATION FOR A CONSERVATION OF HEARING PROGRAM

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TUCSON, ARIZ.

SHORTLY after I came to Tucson, about five years ago, the Arizona State Medical Association asked me to help organize a conservation of hearing program for our state.

I had the privilege of working in the conservation of hearing program in the state of California for several years before that, but the program they had in California was entirely different. There the work was all outlined and set up by the state department. In Arizona we had nothing that was a tie-in at all with our Arizona Medical Association. This offer was a real challenge but after I accepted it, I wondered what I would do and where I would begin.

To understand the dilemma I was in, you will have to know something about the state of Arizona. First of all, it is the youngest state in the Union. Second, it is the fourth largest state in area in our country. Third, we have only about 700,000 people in the entire area. Fourth, over half of all that population is located within two cities, which are Phoenix and Tucson, and they are both located in the southern half of the state.

Then, lastly, the thing we had to consider was that out of the 700,000 people in the entire state—the fourth largest state in the Union—we had only about twenty-five to thirty otologists, and practically all of them lived within the areas of Phoenix and Tucson.

My first step was to contact Dr. Dean M. Lierle at Iowa City and Dr. James M. Brown in Los Angeles. They gave me some good advice about how I should start, who should be included, and what we should do in the way of a conservation of hearing program.

About this time a young lady came into my office one morning, introduced herself as Mrs. Lydia Newton and told me she was at the University that summer and wanted to know if I would help her out with some of the work. She said she had been in Arizona for several months; had been sent there by the National Society for Crippled Children to do a survey on hearing on a statewide basis. About that time all the problems began to take on a different complexion. I said, "Maybe there is something we can do about it, after all."

We began by studying the hearing program outlined by our Academy. We also studied the programs of many of the states throughout the Union. Some of these programs looked awfully good on paper, but we later discovered that they did not function very well as far as the communities were concerned.

Without a well-trained person to do your spade work—and that is to contact the different people throughout the state—do your screening and do the testing of the children, it would be practically impossible for an otologist to find the time required to organize a sound conservation of hearing program on a statewide basis.

Mrs. Newton and I outlined our state program and attempted to adhere as closely as we could to the program as outlined by the Academy. We began

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right at the top. We appointed a state lay committee. We worked down through the different levels, came clear down to the individual and the clinics in the schools.

We were then ready to present this plan to the State Medical Association through our professional committee. I emphasize that professional committee because I am not familiar with some of the programs in other states, but this is a special committee in our State Medical Association. It is made up of the chairmen of the different special committees throughout the state, such as mental hygiene, child and maternal welfare, venereal disease, crippled children, and tuberculosis. It passes recommendations on to our state president.

This enables our president to be constantly aware of all the activity being conducted by the state association and to direct our program accordingly.

This new committee for the conservation of hearing was selected and appointed by the chairman of this professional committee. Our new committee was to consist of three members, Dr. Archie E. Cruthirds, myself, and one other, a man down in the southeastern part of the state, in the Douglas-Bisbee area.

Our committee was to function as an advisory, working committee to the director of our hearing program. At our joint meetings we determined the details of all otological examinations and the place to render treatment.

Then we formulated the plans for our city and county school clinics that were to be developed throughout the state. We made every attempt to keep our program in the public school system. Mrs. Newton has told you how she contacted the boards of education, superintendents, school nurses, school doctors, and any other doctor who might be willing to work in a program of this type. After getting the promise of their cooperation, she explained how she proceeded with the screening of all school children.

The children who had hearing losses were asked to report to the school health department, with their parents, on a specific day. At that time the otologist examined the patient, and the nurse from the student's school recorded the findings and the recommendations. After the examination, she made any further necessary interpretation of hearing loss, the doctor's findings, and urged the parent to carry out the recommendations.

The name of the family physician was obtained in this conference. Incidentally, that was another thing we tried to handle very carefully because Arizona is not specialist-conscious. The majority of the people have a family doctor and they go to him for everything. So, we tried to keep that tie between the family and the family doctor if it was at all possible.

After we examined the youngsters, we mailed a copy of the medical report and the history and the recommendations to the family physician. If the family had no physician and the parents asked for advice on where to have the work done, our director gave them a list of names of otologists that were available and also their addresses and urged the parents to take the child to one of the specialists.

Our director also found out something of the financial status of the family, in this conference. If the family needed financial assistance, they were referred to the county chapter of the Arizona Society for Crippled Children. There a plan was worked out with the medical committee and the society. This was handled confidentially within the committee.

Our children were rechecked at the clinic the following year and a re-evaluation made in the otologic clinic.

This general plan was employed throughout the state, with the exception of the Phoenix area. There it was thought it would work out to better advantage if the otological examinations were scheduled in an otologist's office. As soon as the testing process was completed, the director called each examining otologist and scheduled a certain number of children, with their parents and nurses, in the doctor's office on a given day. By the time Phoenix had an organized, going program, they had the benefit of the growing pains and experimentation that our state program had experienced for the preceding three years throughout the rest of the state.

On the county level—we have only fourteen counties in this great big state—each one is organized from the standpoint of societies. We asked that a doctor be appointed as chairman of conservation of hearing. Of course there were no otologists in some of these counties, but if an otologist was available, he was asked to serve.

Several problems arose because of the fact that most of our otologists were located in metropolitan areas. We had no otologist at all in the northern half of the state. Of course this made it necessary to send some one into the community to work with the local doctors. This was done in Flagstaff and Prescott, which are both in the northern half.

This otologic clinic was arranged by the state director or nurses or the society chairman, and a state director and otologist in one of our two cities of the state would either fly or drive into the areas to help conduct examinations and to make recommendations.

We always invited the local physicians in the town to participate in the clinic, and we tried to make them feel we were there to help them and to help promote what they were doing. We got excellent cooperation from these doctors. Each case was discussed with them or the family physician and arrangements were made for each youngster to have his treatment in that particular locality, if at all possible. If not, arrangements were made to have the youngster sent into the metropolitan area. Transportation to and from this area was provided by the Arizona Society for Crippled Children, Kiwanis clubs and other civic organizations. If we needed any help for further financial assistance, they were also glad to help out.

About this time we again submitted all of our plans and our progress report to the professional committee for approval. This gave our program a healthy beginning and, also, tied the program to the state medical society. We made every effort to tie the program into the Department of Health and the Department of Education, and both of these departments have given us excellent cooperation.

We have conducted special clinics for speech and hearing in conjunction with the summer sessions in our state university at Tucson, and another in our state college located at Flagstaff. The geographical location of the cities—Flagstaff in the northern part and Tucson in the southern part—has facilitated the transportation of children to these special clinics.

Incidentally, the excellent cooperation we received from the university and the college has helped immensely the growth and expansion of our program. It has placed the educational phase of this program in a healthy position.

In the event Arizona ever establishes a medical school—it looks like it is way off in the future, but we hope some day that we will—our speech and hearing clinics can very readily be turned over to and be conducted by the medical group or the medical college.

For the past few years the state medical society, and our state Society for Crippled Children have sponsored a conservation of hearing program at our State Medical Association spring meeting or at the annual meeting of the state Society for Crippled Children. For our first speaker we had Dr. Scott N. Reger of Iowa City, who conducted a panel discussion on conservation of hearing. Those of you who know Scott know that he stimulated a lot of interest. The next year we had Dr. Victor Goodhill from Los Angeles, and last year we had Donald Caziare from the State Department of Health in San Francisco. All of these have helped to stimulate a lot of interest among the lay people as well as among our professional people, and indeed the last group needs a lot of stimulation.

I have attempted to give you a short résumé of our program which has been in operation for only five years. Each state and community will present different problems, but they must be intelligently faced and adequately solved in order to inaugurate a sound conservation of hearing program.

Our Arizona program has grown slowly. We have made a lot of mistakes, but we feel that we have profited by every one of them. We feel we are building a conservation of hearing program that will continue to grow and serve the people of the state of Arizona.

The greatest difficulties we have encountered have been apathy and indifference to and inadequate information about the over-all picture of the conservation of hearing program. Also, a lot of the people, and our doctors as well, felt the whole thing was socialistic.

Our school nurses have been most cooperative but their duties are far too heavy and diverse to carry on the program without any assistance. Volunteer help was enlisted for aiding them in their screening processes. We got these workers through different women's organizations throughout the state—medical auxiliaries, parent-teacher associations, various women's clubs, and the Junior League. They have all come to help us in the different things we wanted to do. In addition, extra help is urgently needed to maintain proper records and to assist in the follow-up of these cases.

We have tried to impress on our doctors the importance of adequate follow-up care in speech and hearing therapy as well as just the plain medical care we can give them.

DESCRIPTION OF THE EDUCATIONAL FOLLOW-UP IN A CONSERVATION OF HEARING PROGRAM

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PHOENIX, ARIZ.

In discussing the educational follow-up in the rehabilitation of the hard of hearing child, my part in the program is to summarize briefly a subject with which the average ear, nose and throat doctor may not be too familiar.

It is sometimes difficult for us to realize the problem of the deaf child. So let us visualize it. Suppose you were the only survivor in an airplane crash in a foreign land. Also, suppose that when you regained consciousness, you found you were unable to hear and that you were surrounded by strange looking people, speaking a strange language you could not understand. How could you make your wants known to them? This parallels the deaf child's situation.

A hearing aid is not always the answer to the correction of a hearing loss. The child needs a training period with an experienced hearing and speech therapist in order that sound may become more meaningful to him, and that is certainly a slow process.

The average ear, nose and throat doctor will follow through on the medical treatment and surgery. He will also see that x-ray and radium treatment is administered in indicated cases, but he may neglect to give the parents constructive advise that will enable them to understand the problems caused by the hearing loss.

It is just as much the ear, nose and throat doctor's responsibility to follow through at least in interest and to support the educational program for the hard of hearing, whether the child needs speech training, auditory training, development of language or the fitting of a hearing aid.

In Arizona we have a few centers that will meet this educational program for the hard of hearing. They are not elaborate and not fitted with expensive equipment like some of the large audiometer centers, but we feel we are making definite progress.

We have two speech and hearing centers in session during the summer: one located at the University of Arizona, in Tucson, in the southern part of the state; the other at Flagstaff in the northern part of the state. These centers provide training not only for children but also for parents and for teachers.

A trained hearing and speech therapist carries out the program to meet the child's individual needs, whether it be speech reading, auditory training, fitting of hearing aids, or all of these.

During the school year there is also a well-organized speech and hearing program in the schools of both Tucson and Phoenix. In Tucson the screened hard of hearing children are brought to a central clinic and examined by the Tucson otologists, whereas in Phoenix the school nurse and parents, with the screened hard of hearing children, are taken in groups of ten or twenty to the individual offices of the Phoenix otologists.

We also have a preschool for deaf children and their mothers in the Phoenix area. This is administered by the Arizona Society for Crippled Children, following a pattern similar to that of the John Tracy Clinic in Los Angeles.

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Most of us now realize that the deaf child entering school is without language, does not know his own name and does not know that the people and objects about him have names. Every possible means must be used to put this deaf child on an equal footing with his brothers and sisters who can hear.

The normal child associates sound with other senses—vision, feeling, taste and smell—in learning to talk and to understand. The hard of hearing child has to make associations through the others senses—sight, feeling, taste and smell—in order to compensate for his hearing loss.

The child with a hearing loss must be taught to observe movements of the mouth, tongue, facial expressions and movements through imitation and constant repetition, with the aid of an understanding parent, a capable therapist, and with the aid of modern equipment, including amplified sound.

The Society for Crippled Children which has charge of the preschool for the deaf emphasizes the instruction of parents so that the child may profit from learning situations in the home as well as in the school.

Slides were shown of some of the activity in the preschool for deaf children. (One showed a group of parents receiving instruction in speech and language development for the deaf child; the parents observing, while the child received the instruction.)

An interest in animals is common to all children, and through animal imitations these children have learned syllables that would have been difficult for them to master through formal instruction. The child demonstrates, "The cow says 'moo.'" The trained ear is used with amplified sound.

Socialization is very important in the development of all preschool children. They have to learn to give and take. Johnny learns to take turns with his teacher. Then it is his turn to make the horse "walk, walk, walk."

Because a deaf child needs more than one avenue of learning, combinations of visual, tactile, and auditory methods are used consistently. The child puts his fingers over the lips to feel the breath sound. The card and the breath sound from the teacher are presented together, showing the visual and also the amplified sound.

In the beginning, each child is given brief periods of individual instruction. As he becomes more familiar with amplified sound, the interest span increases and it is possible for two children to receive instruction together. Deaf children as well as children who hear learn from each other.

Because an abstract concept of numbers often proves a stumbling block in later years, the teacher makes numbers more meaningful to these children through play and through everyday situations. As a result of repetition and numerous experiences, these children match correct numerals with groups of objects. This may seem very simple to us but to these children it is a major performance.

We are recognizing more and more the importance of rhythm in speech. These children babble and pat and march and dance with music. A little boy can distinguish between high and low chords by feeling the vibrations on the piano, with his hand on the piano and his eyes closed. He raises his hand to indicate when he hears the high chord.

Now, as a result of observation, instruction and experience received by parents participating in this program, some of them become quite proficient in giving instruction in lip reading or speech reading, as we call it, thus enabling the clinic to carry a heavier pupil load.

Since World War II, there have been great strides in the development of new equipment. We have found the Speechmaster very valuable in teaching these children the correct position of the tongue, the teeth and the lips.

Even though in Arizona we do not have research centers, we make an effort to take advantage of the newest methods and equipment developed in research centers. We have developed a valuable piece of equipment which we call, until we find a better name, a speech indicator. This is a simple electronic panel of light, but it is very valuable in that it is helpful in indicating the intensity of the voice. It stimulates interest in the use of the voice. It develops breath sounds. It also gives the child a visual register of the rhythmic pattern of speech. The beauty of it is that it brings out these points in a way that is of interest and is understandable to the youngest child.

Most parents stop talking to the child as soon as they know that he is deaf, but, regardless of the degree of his hearing, a deaf person's most valuable asset is his ability to read lips.

As doctors, especially ear, nose and throat doctors, it is often our most difficult task to tell an anxious parent that his child is deaf, and the most constructive advise we can give to this parent is to talk, talk and continue to talk to his deaf child.

"I SCARCE WAS SURE I HEARD YOU"

Saga of the Work in Prevention, Conservation and Rehabilitation by and for the Hard of Hearing

EDMUND PRINCE FOWLER, SR., M.D.
NEW YORK, N. Y.
BY INVITATION

"I SCARE was sure I heard you." This line of Poe's was chosen for the title of my address because it well serves to distinguish the state of the hard of hearing from that of the deaf. At this time we are concerned primarily with the hard of hearing; the deaf and the schools for the deaf will be mentioned only incidentally.

It is an honor to be chosen by the Otosclerosis Study Group to tell the story of the birth and growth of the work of the organizations for the hard of hearing. I trust that my treatment of the subject will convey to you more than a tale of struggle and mounting success; rather a story of valiant endeavor by the hard of hearing in behalf of the hard of hearing, of their contributions to otology by teaching, research, understanding and, above all, by constructive thinking and example.

In order to better understand some of the problems involved, a few words should be said about the mysteries of the phylogenesis of the ear, hearing in general, and the philosophy of rehabilitation or adjustment to deafness.

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Hearing is in essence a sensation of shearing touch or pressure. No matter how lowly, all animals, and in a sense even some plants, possess an ability to hear if by hearing is implied an ability to react to vibrations in their immediate environment. However, only terrestrial fauna possess organs specifically developed to hear and differentiate air-borne sounds.

Countless eons ago our prehistoric ancestors living in the sea and in the vaporous or steaming ooze of the tidal wash along its shores and indentations, or in inland water pools large and small, slowly evolved organs of hearing from what at first was a general surface or skin touch apparatus. This eventually became localized and transformed into a small organ on the surface of the body. Sometimes this organ (this primitive ear) was on the head, sometimes on the back or tail, and sometimes, as in certain insects, on the feet or other parts of the body. (Reminds one of the places recommended for certain hearing aids, doesn't it?)

The structures forming the ear of man and the higher vertebrates have been traced phylogenetically, with large gaps, down to the primitive forms, as in the jellyfish. These structures are called "anlages." They were at first useful only for sensing underwater vibrations, and so our primordial progenitors, like the present-day fishes, lived in a relatively silent world although they sensed pressure variations and, in certain species, possessed some discrimination of vibration. Living under water, they could not sense air-borne sounds. To quote again from Edgar Allen Poe, "The quiet was unbroken, and the silence gave no token"—no token of the presence of terrestrial friend, foe, or food. There was no sound, if by sound we mean audible vibration, because air-borne sounds are reflected from the surface of water; they do not reach the depths. With the slow lapse of ages as the seas receded and land masses and terrestrial animals appeared, more predatory foes with or without legs or wings were on the prowl, and more and more it became necessary for survival and for the perpetuation of the species to hear air-borne sounds in order to avoid danger, to sense the presence and localization of prey and to hear the mating call of the opposite sex. (Some cynic might call the last named "courting" rather than "avoiding" danger!) (No double pun intended.)

Few individuals or species survived without possessing, first, good sensation for water-borne sounds and, eventually, good hearing for earth-borne and air-borne sounds. Good hearing was a large factor in the survival of the fittest although the other senses played important roles. (Even the so-called lower animals may be said to have hearing for a kind of primitive speech although the sounds they produce and hear may be beyond the range of the human ear.) In many terrestrial animals, including mankind, although eyesight, touch and smell complicate the picture, the love song above all still is heard throughout the land, mankind seemingly being the only animal to deliberately distort it into the dissonance of "bee-boop," or "bee-bo" or be-something else equally disagreeable to a sensitive ear.

It is usually said that in man there are five senses—sight, smell, taste, touch and hearing—all of which may be useful in certain types of communication. To these I would add those of body coordination and orientation in space and movement. Two of the sense organs, those of equilibration and of hearing, are housed in the chambered capsule of the inner ear, and these two even in the higher forms are still basically similar in function. One senses motion (and absence of motion) of the head and body, the other senses motion (and absence of

motion) in the environment; whether it is water, earth or air. When motions in the environment cause a sensation of sound, they are called sound waves or vibrations.

All the senses with the exception of hearing and sight are concerned mostly with the immediate environment—in other words, more with near than with far factors. However, while the eyes are closed, sight ceases, and when the ears are blocked, sounds are less readily detected.

Hearing is a social function. It is a very important social function but it is not an absolute social necessity. It is not a guarantee of happiness; many flourish without it, many wither who possess it. Of course absence of both speech and hearing is much more distressing than a loss of either alone.

There are signs that in the not so distant future, because of lessening importance for survival, both speech and hearing in man may gradually deteriorate. Will this be good or bad? Who knows?

More than the ear is necessary for good hearing because for all sense organs there is a central (or psychic) apparatus without which the sense in question produces only an automatic, reflex, low level, or unconscious response valuable for alarm reactions directly to the muscles of the body but useless for fine discriminatory functioning. When you jump at a loud noise even before you are aware you have heard it, that is an example of an alarm reaction.

To understand speech or other sounds fully we must employ more than our outer, middle and inner ears, we must possess and use both central and peripheral analysis, we must use our brains as well as our ears. This is one reason why so-called exact or observational research is unable to give us all the answers. An absolute value cannot be assigned to any biologic measurement. There is no standard pattern of response, there is only a range of variables. Psychic response is often unpredictable and unstable. The old saying, "Grandma can hear when she wants to hear," still often holds true. Remember that one may consciously and deliberately ignore a stimulus. I once knew an old rascal who would not admit he heard a sound unless a pretty girl put her lips close against his ear. He got a great kick out of this. You see there are compensations even for severe deafness if you have a little imagination and use it.

The hard of hearing have always been aware of the varying and unpredictable responses to sound stimuli in deafened persons. They have demonstrated throughout the years that in many instances mere amplification of sound was not enough to enable them to hear. They have in their organizations provided the people, the living laboratories, in which research has resulted in a better understanding of the problems involved. They have been living examples of the effect upon the hearing of the many disorders which affect the mechanical and neural mechanisms of the ear.

The hard of hearing people themselves have been the pioneers in what is now called "audiology." From the first they have spread the gospel of early detection of deafness in the adult and especially in the child; they have stressed the importance of frank acknowledgment, and early diagnosis. Without diagnosis you can arrive at no prognosis, and without prognosis you can make no satisfactory plan for management and treatment.

Incidentally some people forget that by diagnosis the otologist means determining which of several diseases or disorders is causing the symptoms and the pathology, not simply the amount and pattern of the hearing loss. It is stressed that even early detection and diagnosis and medical and surgical treatment and

follow-up are not enough. Continuing research is imperative.

Before the twentieth century there were probably as many deafened people in the world in proportion to the population as today, but only the very deaf, the "stone deaf," were really cared for. The institutions of those days taught, and some even today teach, only the sign language and not phonetic speech, nor what is today usually called "lip reading." This tended to limit the social world of the severely deafened to their schoolmates and those few others who knew the sign language. The only efficient means for aiding the hearing was some form of speaking tube or trumpet which although it did not amplify the voice, did enable the voice of the speaker to be channeled directly into the ear of the hearer. It had other valuable assets: it excluded unwanted and confusing sounds, it was rugged and it had no movable or easily breakable parts, it was usable in all forms of deafness unless there was a total inability to hear speech. The speaking trumpet is still a useful instrument in very severe deafness. It is inadequate for distant speech. It is unsightly and a nuisance. Grandma usually for years could hear without it if she really tried, and so she was wont to use her cupped hand and put off using the trumpet, or any other hearing aid, until her family and friends almost lost their voices trying to make her hear them. For years Grandma could be a trial to her friends and to herself.

But Grandmas and younger people, too, need not, and many did not, permit deafness to upset their lives. In the organizations for the hard of hearing we proved that many people with severe deafness could become adjusted to their deafness and even better adjusted to life than millions who possessed perfect hearing. Every man must learn to live with his limitations.

It may be said that "he has not lived who has not suffered pain, sorrow, and frustration," and learned how to live with himself. Otherwise he knows not the destiny of man. He is not mature. He is not grown up. The hard of hearing have suffered, they have lived and many have grown up. Those who have not may at any time do so by facing facts and availing themselves of the aids now available.

Even before the turn of the century a few brave and venturesome souls began to gather in little groups here and there in the larger cities, some purely for human contacts, some so that they could exchange opinions concerning their deafness and head noises, some so that they could have what they thought would be a receptive audience for their emotional outbursts, and at first a few, a very few, mature and earnest men and women who wanted to do a job to help the hard of hearing people to help themselves. Otherwise there was little interest in these matters in the first decade of the twentieth century, although other handicaps received much attention, particularly blindness, paralysis and physical deformities.

The deafened people, whether speechless or not, were listed under the category of "deaf and dumb." "Dumb" connotating not only inability to hear and speak but stupidity. In 1938 under the urging of the American Hearing Society the United States Civil Service was persuaded to change its designation from "deaf mutes" to "the hard of hearing." It is clear who had been the "deafer and the dumber."

The deaf were often the butt of crude and cruel humor off and on the stage and this was resented by some deafened people, thus maybe starting the trend which now would taboo any humorous references to what is sometimes thought to be amusing characteristics of the Irishman, the Negro, the min-

ister or priest, the Quaker, the Shaker, the Jew or the Christian. Protests were made even in the silent-film days in regard to certain jokes about deafness. What a mistake this was. The deafened people themselves found out that the only one who approaches emotional stability is he who can laugh at himself, and today in the organizations for the hard of hearing they put on skits illustrating the very funny things that happen because of deafness, and the people who enjoy these the most are the hard of hearing themselves. This is one of the things I tell my patients.

One of the greatest deterents to the formation of organizations for the hard of hearing has been a dislike, aversion, timidity or cowardice on the part of some hard of hearing people and their families, and some of their friends most able to help financially, to be publicly associated with the hard of hearing. They wanted to hide their deafness and the deafness of their relatives and they imagined they could do this because they wishfully tried to think that deafness is invisible. As a matter of fact deafness is invisible only so long as nobody speaks, expects an answer, and either receives none or is baffled by one that is based upon faulty hearing. It is frequently even more apparent than blindness. The minute you speak to me and it is evident that "I scare was sure I heard you," my deafness is no longer invisible. Why attempt the impossible? This philosophy is taught best by those who are hard of hearing, they are living examples of its efficacy. Some hearing aid manufacturers and salesmen have been guilty of perpetuating the understandable desire of the timid and ill-adjusted to hide deafness by concealing the hearing aid by overemphasizing in their advertising what they call "invisible hearing aids." This type of advertising should be discontinued.

Many deafened people feel that their associates are irritated by their deafness, but the real reason why friends and relatives are irritated by some hard of hearing people is not because they do not hear well, but because of their often strange overemotional, even hysterical reactions and behavior. The teachers of the hard of hearing in the chapters of the American Hearing Society were the first to fully understand and correct such reactions and to inculcate a proper philosophy in their pupils. One reason why the most successful teachers of the hard of hearing are usually hard of hearing themselves is that they are a living unanswerable argument to those who say that all of the emotional and other troubles of the hard of hearing are caused by the deafness. These teachers are far better adjusted to life and its inevitable emotional episodes than many of the teachers with perfect hearing holding forth in our schools. They have profited by a proper understanding of their possibilities and limitations. It is an interesting fact that in some people deafness serves more as a stabilizing than an upsetting factor. The motto of such people is "Adapt to deafness, do not fight it." Do not fight your hearing aid, learn how to use it.

A proper philosophy is important not only for a satisfactory life but because it lessens excessive emotional reactions, and protects the ear from neurovascular anoxias. It is therefore an important prophylaxis in several forms of deafness.

The pioneer in organizing the hard of hearing in this hemisphere was Edward Nitchie who, beginning in 1903, gradually built up a group which in 1911 was called "The Service League," and in 1912 was incorporated. It had one paid social worker, Miss Anetta W. Peck. It was wholly supported by volunteer contributions and membership dues.

This started the ball rolling and attracted the cooperation of a few otologists.

Within two years the Nitchie group expanded and was renamed and chartered as "The New York League for the Hard of Hearing." At first it had only one paid worker, twenty volunteer workers and a capital of $400, a blackboard, some "hand-me-down" furniture and worn packs of cards. From the first the plan was not only to provide meeting places for the hard of hearing but to improve their social happinesss and economic status and their opportunities for self-advancement and employment, to investigate and advise in regard to the proper choice and use of hearing aids (1922), to protect the hard of hearing from being fooled by misleading advertising, to steer them away from quackery of all kinds. Surveys were instituted* for the purpose of detecting and diagnosing hearing losses in children, and by medical follow-ups and teaching to help the hard of hearing children to continue in the regular schools instead of being segregated in the schools for the deaf. They also attempted to educate not only the children but their parents, their school teachers and the general public in hygiene and common sense measures for the prevention of deafness. These things it did to the best of its ability as did all of the organizations for the hard of hearing subsequently organized.†

During World War I the larger organizations grew to real professional status and the recruiting officers for the armed services referred to them men rejected because of hearing defects. They arranged lip reading scholarships and vocational readjustments for hard of hearing soldiers and sailors, and the experience gained proved invaluable a quarter of a century later in and following World War II. Now thirty odd years later several societies, some "speech and hearing clinics" and commercial "hearing aid clinics," are attempting to organize what they call similar services, thus far with but little success from the viewpoint of the hard of hearing people. Some so-called clinics not sponsored or approved by the American Hearing Society seem more interested in selling hearing aids than in really helping the hard of hearing. This should be stopped.

In 1919 Dr. Wendell C. Philips, then President of the New York League for the Hard of Hearing, brought the groups in the various cities together to form the American Federation of Organizations for the Hard of Hearing. Today it is the American Hearing Society.

Starting with maybe only a room for meetings many of the Chapters soon moved to larger quarters as they expanded, and some purchased their own quarters. With the advent of radio in the early 1920's headphone attachments were obtained for group teaching and group enjoyment of these devices. In 1920 free lip-reading classes were instituted in the schools in some of the larger cities, and in New York City alone over 11,000 requests were received from hard of hearing persons for advice and assistance in their problems, which varied from the selection of hearing aids to the advisability of undergoing eustachian tube manipulations, so-called cures, magic ear oils, and fakes

* The first audiometric and medical survey of the hearing of school children was made in 1926 with the cooperation of the Bell Telephone Laboratories. The data obtained from this survey still are widely quoted. Because of the increase in population the then calculated "3,000,000 deafened school children" in the United States is still near the truth even though the 6 decibel criteria for hard of hearing is now increased to 9 decibels.

† I do not wish to overstress the New York League for the Hard of Hearing, but being active in it for over thirty years and its president for eight years, it is difficult not to bring it frequently into the picture. Limitations in space and time prevent me from detailing the work of all of our fine chapters and from giving you the names of their valiant pioneers.

of various kinds. Several of the chapters even employed most efficient "Dorothy Dix's" to advise the lovelorn. Groups for the young and for old timers were organized, and all made use of the recreational and social facilities. The chapters grew, and many exhibited several types of "ear phones." No charge was made for any of their services.

The chapters of the American Hearing Society throughout the country did more to popularize and expand the use of the hearing aid than any other agencies, and the members and professional staffs took delight in wearing their aids so that all and sundry could see them and profit thereby. Many prominent people came to the League and aided in these matters. I wonder if the hearing aid manufacturers understand and appreciate this? Some of them criticize us because none of our organizations exhibit every type of hearing aid in their testing rooms. I ask them, "Does any manufacturer or dealer have every type of hearing aid in his salesrooms? Does he test people in his salesrooms with any aid except his own?" For obvious reasons the answer is, "He does not."

Many of our chapters have exhibited many hearing aids, some as many as twelve or more, all approved by the American Medical Association Committee on Audiometers and Hearing Aids, and this is more than is necessary for a proper selection.

The organizations for the hard of hearing contributed much to the improvement of the odd looking contraptions that were manufactured in the early days, and to the reliability, standardization and diminution in the size of batteries. In the beginning the hearing aid was only a carbon microphone, a telephone placed in or over the ear and heavy dry batteries carried in a pocket clipped to the clothing or slung about the neck. This aid did magnify sound, but if it was not properly adjusted, it made life unbearable by the loud "frying" and "sizzling" and other noises it produced. Moreover, some of the original aids were as large or larger than present-day television sets. An early so-called portable model weighed about seven pounds. However, these aids were avidly adopted by the different hard of hearing organizations for use in their speech teaching, speech reading and speech hearing clinics, for the rehabilitation and re-education not *of the hearing*, but *of the hard of hearing person.*

In 1926 the New York League for the Hard of Hearing established under my direction a clinic for the prevention of deafness at the Manhattan Eye, Ear and Throat Hospital. This was the first of its kind and I believe it set a pattern for the development of other clinics in other boroughs and throughout the country. In this clinic every patient was given a complete otological examination, including audiometric testing in a sound-proofed room, stereoscopic x-rays of the petrous bone, differential blood count and analysis, and in many instances determination of the basal metabolic rate. Every item was listed and catalogued under the various categories of diagnosis. These records are still available. Today there are other clinics especially interested in prevention and rehabilitation, but without the assistance of properly functioning employment services and of classes for children and their parents, as well as for adults and the aged, they will not be able to do the work established and still being carried out by many chapters of the American Hearing Society. It is sometimes forgotten that the most valuable piece of clinical equipment is a trained and understanding operator not a piece of electrical or mechanical apparatus.

In cooperation with Teachers College, Columbia University, the staff of Chapter I, American Hearing Society, gave courses on the problems of the

hard of hearing and on methods of teaching lip reading and speech training to deafened children. A text was prepared for teachers of the hard of hearing. All were under the supervision and active participation of Miss Estelle E. Samuelson, who has for many years been secretary of the New York League. Miss Samuelson wrote the first textbook for training of children, and Mrs. Konnei wrote a book on auditory training, "Learning to Look and Listen," and a book for the children themselves as well as for their parents, "Tim and His Hearing Aid."

During the "Hoover-Roosevelt depression era" there was widespread unemployment. The American Hearing Society, under the inspired guidance of its officers and loyal staff headed by its Executive Secretary, our loved and lovely Betty Wright, valiantly persisted in spite of cuts in salaries and hardships among its workers and members. Jobs were obtained for hard of hearing workers. A large number of other welfare agencies failed to survive the depression, but no chapter of the American Hearing Society died. One of the most generous member contributors to the cause was Mr. Starling W. Childs. Mr. Childs was also the largest contributor to the Central Bureau of Research, whose trustees are the custodians of the Research Fund of the American Otological Society, Inc.

The larger chapters set an example for the smaller organizations by establishing funds for supplying free hearing aids for the indigent, and revolving funds for helping others to purchase aids. Many hearing aid manufacturers sold us aids at cost, and some from time to time donated aids. The American Hearing Society was the pioneer in setting up machinery for yearly examination and follow-up and auditory training projects for children so that the residual hearing of even the severely deafened might be utilized to enable them to hold their own with normally hearing children in school, at play and in the world. It is no longer unusual to see children happily wearing hearing aids. More should do so.

Nursery schools and classes for the instruction of the parents were established and are now becoming widespread. Without parent cooperation in management and teaching, many deafened children flounder and become more and more emotionally unstable, as do their parents.

The American Hearing Society received national recognition and was asked to serve at the White House Conference on Child Health and Protection. The New York League prepared forms and cooperated with the Federal Government and the Board of Education in training the technical staff of the WPA project for testing the hearing of school children in the New York City schools. Chapters in other parts of the country rendered similar services.

In 1938, 1939 and 1940 several members of Chapter I served on a New York State Legislative Commission to study facilities for the hard of hearing. I believe this was the first commission of its kind in the country. In cooperation with the Welfare Council, information service was expanded and the New York League became a member of the Greater New York Fund. Other organizations of the American Hearing Society in increasing numbers have become members of the Community Chest funds in their areas. Under the presidency of Dr. C. Stewart Nash the number of chapters participating increased to fifty. Now, I understand, they number seventy.

During the late 30's and since, due largely to developments in electronics in World War II, hearing aids have been greatly improved and streamlined. The hearing aid and vocational consultation

services were swamped, and even today bookings are made six months or more in advance.

Before the Veterans Administration program was operative, a full-time service center was set up to meet the needs of the returning war deafened veterans of World War II. This was known as the "Veterans Sounding Post." It was established by the New York League in memory of the late Lt. Lester N. Hofheimer, USAF, a casualty in India in 1943. It provided psychologic case work, home adjustment, lip reading and speech instruction, hearing aid service, recreational and employment services for all branches of the armed forces. The Caduceus Post, 818 American Legion, has made yearly contributions to this activity.

The American Hearing Society is the voice of the hard of hearing people of America. It now has 117 chapters and over 10,000 members. (The goal should be over 100,000.) It has pioneered in all of the services I have mentioned, and in addition in organizing community supporting groups, in developing the screening phonograph audiometer technic, in promoting state and national legislation for physically handicapped children, and in conservation of hearing programs in schools, National Hearing Week, the publication of a hearing journal, *Hearing News*, and hundreds of informative phamphlets.

Our statistics show that deafness affects more than twice as many persons as cancer, heart disease, tuberculosis and infantile paralysis combined yet fewer people join and still fewer contribute to the organizations for the hard of hearing than to these other health agencies.

Although large numbers of social workers, school administrators, teachers, health and rehabilitation officers, speech and hearing therapists are now among its members in many locations, otologists in sufficient numbers have been conspicuous by their absence. This is unfortunate because every organization for the hard of hearing needs their guidance. They need otologists who are cognizant not only of the diseases and disorders of the ear but of the social and psychologic repercussions often involved in deafness. If otologists neglect this field of endeavor they will find that the so-called audiologist, the deaf cure and hearing restoration practitioners will be taking over. In fact, it would almost seem that some otologists think that deafness is not a primary concern of otology. Well, if correction and rehabilitation services are not problems of the otologist, then correction and rehabilitation are not problems of the ophthalmologist. The belief that less can be done for deafness than for blindness is a myth. In many instances much more can be done to improve the hearing than can be done to improve the eyesight. No eyeglasses can bring back the sight in cases of eye obstruction. In the ear, obstructive deafness is easily overcome by hearing aids and, in certain forms, by operative procedures. Let's keep this straight.

The hard of hearing are vitally concerned with the inheritance of deafness. Much specious preaching has been heard on this subject. It is not generally realized that human genetics is far from being an exact science, and therefore no one should try to play God. However, the chapters of the American Hearing Society have cooperated with the geneticists and otologists and have provided many cases for study in heredity. The majority of the identical twins under my observation for the past ten years have been referred from two or three chapters of the American Hearing Society. We have been concerned with factors seemingly involved in the heredity of deafness and in the activation

and exacerbation of the lesions of otosclerosis.

One of our problems has been the unwarranted discrimination against the hard of hearing in industry and in certain professions. It required many years of persuasion to stop certain boards of education from dismissing competent teachers just because their threshold audiograms were not up to some arbitrary standard. In some instances the wearing of a hearing aid was used as an excuse for dismissal. Fortunately such practices are lessening; but at the moment the scarcity of first class teachers in our schools will help terminate them because it will demonstrate how efficient the wearer of a hearing aid can be.

Age has been used as a criteron of performance of the brains, the muscles, and the ears, but not so much of the eyes. It has recently been proposed that anyone 50 years of age or older should suffer a deduction from his compensation for hearing loss because on the average those over 50 do have some loss in high tones. There are so many variations and exceptions that such an arbitrary procedure is obviously wrong. What has the average got to do with it? We are interested in the individual performance, not in an average at a given age. Do not let the physicists and experimental psychologists tell us what our patients should hear. The ultimate criterion for evaluating hearing capacity is what our patients do hear, as determined by our clinical observations and the audiometric measurements checked with tests for speech hearing at different levels.

During the past thirty-five years there has been an increment of understanding of the problems of deafness, of its etiology, pathology, detection and alleviation. Many have contributed in many fields of endeavor and it is not entirely fortuitous that these years have coincided with the growth and work of the organizations for the hard of hearing.

Under the presidency of Dr. Thomas L. Tolan the American Hearing Society is now stressing courses for training the executive directors and executive secretaries of chapters. Research projects and a cooperative plan for financing the national and chapter organizations are in competent hands.

I close with an appeal to the otologists, the audiologists, the hearing aid manufacturers and salesmen, to all good doctors and to all good people everywhere—do not belittle the American Hearing Society. Instead, cooperate with it and help it wholeheartedly, because there is no better way to serve these courageous people who have already helped themselves and the hard of hearing everywhere so much.

DOES OTOSCLEROSIS CAUSE COCHLEAR NERVE DEGENERATION?

STACY R. GUILD, PH.D.
BALTIMORE, MD.
BY INVITATION

WHEN Dr. Meltzer, the President of the Otosclerosis Study Group, asked me to speak on this subject, he knew of my belief and of the reasons for it. They had been published, following their presentation at the 1943 meeting of this Study Group. Since the publication of that paper, entitled Histologic Otosclerosis (Ann. Otol., Rhin. & Laryng., 53:246-266 (June) 1944), nothing has occurred to cause me to revise my views with respect to the effect of otosclerotic lesions on the nerve of hearing. The present paper is, therefore, of necessity, mostly a repetition.

Dr. Meltzer hopes that the topic will call forth a lively discussion; so do I. Some of the discussants will doubtless disagree with my views on the topic. If they do, and if they have evidence to support their conclusions, I shall be delighted. But I issue the warning now that any arguments based solely on hearing tests will be answered with the query: What is the evidence for the correctness of the interpretation made?

The phrase "nerve degeneration secondary to otosclerosis" certainly is a convenient way by which to convey to other otologists the general picture of a patient's hearing tests; I admit to using it myself in conversation. But if the phrase is not properly descriptive of the underlying organic condition, the interpretative prognoses may be incorrect and its use should be discontinued.

From the Johns Hopkins University School of Medicine.

Presented at the meeting of the Otosclerosis Study Group, Oct. 11, 1952, Chicago, Ill.

There is no doubt whatsoever that cochlear nerve degeneration occurs in some persons who have otosclerosis; also, there is no doubt that cochlear nerve degeneration occurs in some persons who do not have otosclerosis. These are facts for which there is good histopathologic evidence. The facts can be confirmed by anyone who is able to recognize the lesions of nerve atrophy and of otosclerosis on examination of sections with a microscope. The real question is whether or not the lesions are causally related.

All attempts to reword the parts of the previous paper, Histologic Otosclerosis, that concern the question of cochlear nerve degeneration secondary to an otosclerotic lesion have been less satisfactory to me than is the published wording. I shall therefore quote extensively from what was presented to this Study Group in 1943. On that occasion, because of interest in other aspects of that paper, the part of it to be repeated here did not receive much, if any, attention in the discussion that followed the presentation. Dr. Meltzer and I hope that the situation this evening will be different.

[In the verbal presentation, the material on five pages (257 to 261, inclusive) of the earlier paper was quoted in extenso. Readers are referred to this published material. Ten photomicrographs, in the form of lantern slides, were also shown.]

The evidence presented in the quotations was summarized as follows, in the earlier paper (Conclusion No. 3, on page 266):

Atrophy of cochlear nerve fibers or of the organ of Corti does not occur more often in ears with otosclerotic areas than in ears free from otosclerosis. When atrophy does occur in otosclerotic ears it is usually limited to the basal turn and differs in no way from the nerve and end-organ atrophy that is often seen in the sections of ears without otosclerosis. The etiology of cochlear atrophy in ears without otosclerosis cannot, in most cases, be established, and it therefore does not seem logical to attribute to otosclerosis all the cochlear atrophy found in otosclerotic ears.

As of today, the histopathologic evidence still warrants the earlier written conclusion just quoted. To the question of whether otosclerosis causes degeneration of the cochlear nerve my answer therefore remains, No.

In closing, I wish to mention the question that logically follows the negative conclusion. The question is: Can a lesion other than "nerve degeneration" cause the clinical picture that has customarily been interpreted as "nerve degeneration secondary to otosclerosis"? To this question I believe the answer may prove to be, Yes.

Physiologic dysfunction of structures normal in histologic appearance cannot be excluded as a hypothetical explanation for the impairments of hearing usually interpreted as "nerve degeneration." Such an explanation is unsatisfactory, to say the least. Personally I prefer to speculate that the true answer may be related to morphologic differences in ankylosis of the stapedial foot plate, such as location of the ankylosed segment or segments, or rigidity of the abnormal connection. In other words, perhaps the effect on hearing of ankylotic immobilization of one part of the foot plate is different than the effect of immobilization of another part; and perhaps the effect of one combination of two or more areas of ankylosis differs from that of some other combination of multiple osseous connections between the foot plate of the stapes and the margin of the oval window. From the evidence that is now available, the possibility cannot be excluded that certain forms of ankylosis may impair the transmission of high tones more than of low tones and also interfere with the transmission of "bone-conducted" sound to the intralabyrinthine fluids.

Until the above-suggested possibility and all other possible explanations of the clinical picture under discussion can be ruled out, my attitude is that use of the phrase "nerve degeneration secondary to otosclerosis" is detrimental to the advance of knowledge and should be discontinued. Certainly the continued use of the phrase tends strongly to discourage seeking for other possible causes of the clinical picture thus interpreted.

DISCUSSION

DOROTHY WOLFF, Ph.D., New York, N. Y. (by invitation): Two or three years ago I spoke before this group in regard to the microscopic picture of otosclerosis. I attempted to show at that time that we had inherited a multiple picture of otosclerosis, and I tried to stress the fact that otosclerosis has no single etiologic factor.

I should like to take issue with Dr. Guild's statement that in his case with tumor of the pituitary, there could be no relation to the otosclerotic picture. I think that it very likely had a very important relationship. I suppose it would be impossible now to prove my point from the Johns Hopkins' autopsy report, and yet it might be possible. The pituitary tumor would undoubtedly alter the association with the adrenal cortex hormones which would also control the metabolism for calcium and therefore might have important bearing on bone pathology. Perhaps in the Hopkins files there are x-rays indicating a generalized bone pathology. We know that otosclerosis may be part of Paget's disease, of gargoylism or of blue sclera. When bone pathology from whatever cause blocks either round or oval window, the clinical diagnosis of otosclerosis may be given.

I will now show you some of the different types of histologic patterns producing otosclerosis.

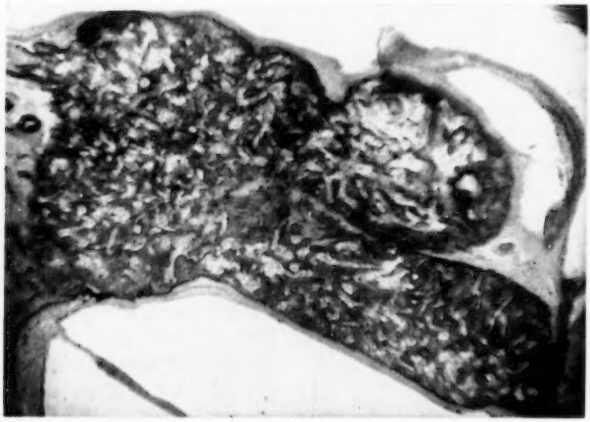

FIG. 1—Otosclerosis taking the form of a large tumor (angioma of the bone). It extends from the promontory over the oval window. Note the thickened and ankylosed foot plate of the stapes. This is a case of Dr. Julius Lempert's.

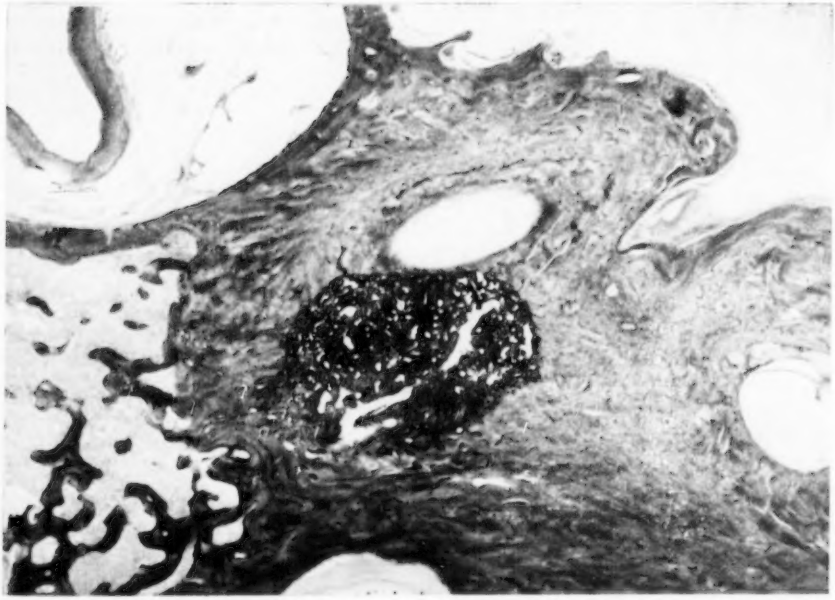

FIG. 2—Radiating paths of the lesion extend along vascular channels. Note the course of a large atrophic vessel through the center of the diseased area. This type of degeneration sets the stage for osteoporosis (Washington University Collection, no. 8630)

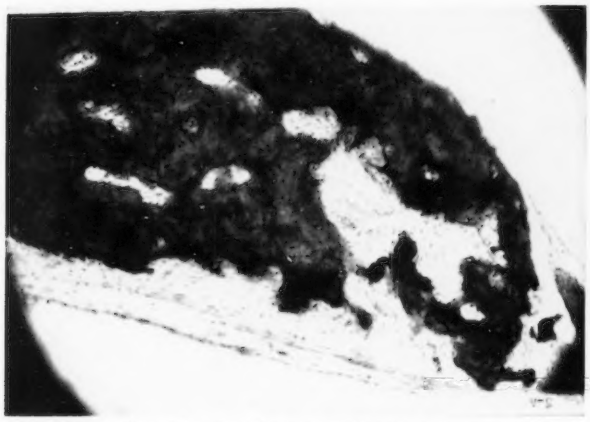

FIG. 3. Foot plate of stapes showing osteofibrotic replacement of bone. The condition was bilateral. Ankylosis of stapes was present in the opposite ear, the fenestrated ear. (Lempert Institute, no. 5)

Figure 1 is from a case of Dr. Lempert's in which there is quite obviously, it seems to us, an angioma of the bone. Dr. Lempert has long thought of otosclerosis as being a tumor, and his idea is certainly justified in this example. This tumorlike growth is coming down from the promontory and, as you see, there is a very thickened foot plate of the stapes with ankylosis at both anterior and posterior borders. So that is one microscopic picture of otosclerosis—a hemangioma of the bone.

I collected a specimen while I was at Washington University in St. Louis (fig. 2). It shows an example of an otosclerotic lesion that occurs neither at the round window nor the oval window but bordering the cochlea between the two windows; consequently there were no clinical symptoms. You see the characteristic clearly demarcated bone, with the advance of the disease going along haversian canals. But what I wish to point out particularly is the large vascular channel traversing the center of this lesion. Obvious atrophy of the wall of this vessel has occurred. Undoubtedly after this lesion was built up by hypervascular activity, an embolus or something occurred—we cannot say what—which stopped the flow of blood to the region. The result is this osteoporotic picture. Cochlear nerve degeneration as well as atrophy of stria vascularis was present in this case.

Another kind of lesion occurred in the foot plate of the stapes in a patient with bilateral otosclerosis (fig. 3.). The Lempert Institute received both temporal bones from an outside source. Obviously you have in this foot plate of the stapes, a type of bone pathology that is expressed as osteofibrosis. This was true throughout the lesions in both ears; an obviously different microscopic picture from either of the two preceding cases. If I may, as the saying is, stick my neck out on this, Dr. Guild, I would suppose that this particular type of lesion would not show nerve degeneration whereas a vascular lesion might (or might not) show nerve degeneration. In other words I would classify this case solely as one of bone change. In this case the nerve was degenerated, but death followed temporal lobe abscess, and a labyrinthitis was present following a fenestration operation four months previously.

An otosclerotic lesion without ankylosis of the foot plate is shown in figure 4. The otosclerosis was bilateral.

There was apparent in the internal auditory meatus of this individual an area of atrophy of the vestibular ganglion (fig. 5). When you examine the area in detail you find that this was due to obliterative arteritis. Whether it was periarteritis or endarteritis, we cannot say now, for histologically they cannot be distinguished after the diseases have progressed beyond a certain point. It is interesting to know that otosclerosis was not diagnosed in this case and to see that there was no an-

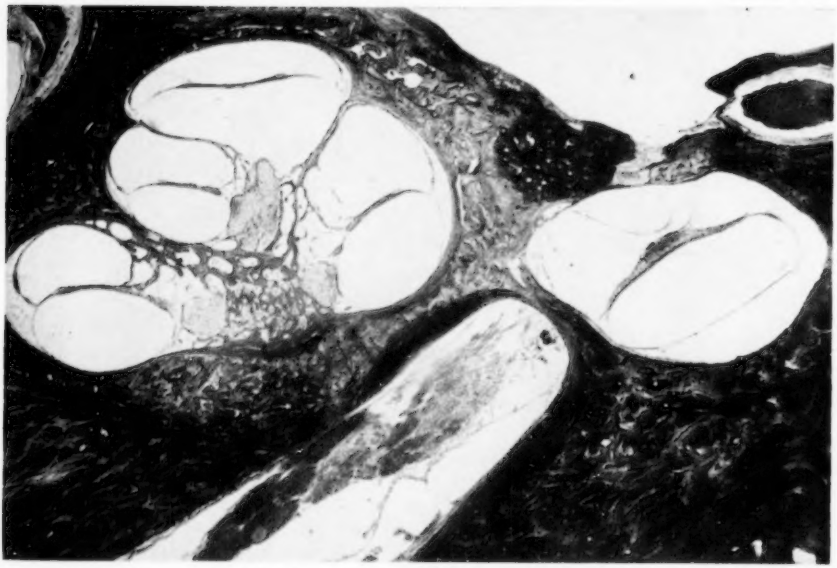

FIG. 4.—Obliterative periarteritis in the vestibular ganglion in a case of bilateral otosclerosis and hydrops of the labyrinth. (Washington University Collection, no. 5178 right)

kylosis of the stapes. The diagnosis of hydrops was made in this case, which was substantiated by the microscopic sections. Dr. Horn, the clinician, said that there seemed to be something else besides the simple hydrops. We can now realize that the obliterative arteritis might easily have been occurring at higher levels in the blood supply of the brain and thus confused the clinical picture. Periarteritis nodosa was not being diagnosed by the general pathologist at the time this specimen was collected by me. It is now suspected that periarteritis nodosa takes origin from lesions of the adrenal cortex. Others ascribe its origin to continuous exposure to damaging vibratory phenomena. It probably represents the organism's reaction to peripheral shock of whatever nature, be it chemical (hormonal) or mechanical (vibratory). This patient was a dentist, probably working in the era when dental drills did not have proper protective sleeves.

We observed a pair of ossicles that were removed from a case with a clinical diagnosis of otosclerosis (fig. 6). You see extensive ankylosis of the malleus and incus, and it was much more extensive than is evident from this single picture. But you will also notice that these bones have widespread osteoporotic lesions. Let us examine the cause of this osteoporotic type of lesion.

Figure 7 is a high power view of the vessels seen in figure 6. This individual was undergoing a state of obliterative endarteritis. Some vessels were completely occluded by proliferating endothelium; others showed only partial occlusion, and still others were completely obliterated. These ossicles were sent to the Lempert Institute by Dr. MacCready. They were taken during a fenestration operation from a patient diagnosed as having otosclerosis.

Figures 8 and 9 taken during the fenestration operation, show the stapes from one of Dr. Lempert's cases having a diagnosis of otosclerosis. You will notice that in the posterior crus of the stapes there is a type of bony lesion that one might almost call osteoporosis and that the descending process of the incus is ankylosed to the head of the stapes. You will see that a blood vessel which runs from the descending process of the incus to and across the head of stapes is actually pressed shut by exudation of globules. We do not know the nature of this globular substance, whether it is lipoid or steroid; in some cases, at least, it may be of gaseous nature (carbon

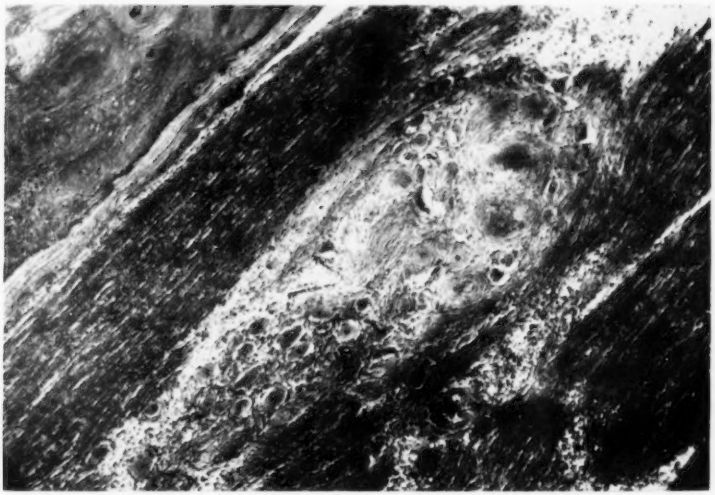

FIG. 5—Detail showing obliterative periarteritis in the vestibular ganglion in a case of hydrops of the labyrinth and bilateral otosclerosis. History of disorientation and possible Ménière's disease. (Washington University Collection, no. 5178 right)

dioxide). But we do see these substances aligned in rows of clear globules within blood vessels or being exuded through the walls of vessels. When this latter condition exists, decalcification of the surrounding bone is seen. When the blood supply is pinched shut, osteoporotic bone is observed. Thus we have shown three ways whereby osteoporotic bone may result.

FIG. 6—Ankylosis of the malleoincudal joint in a case of bilateral otosclerosis of man 36 years old. See figure 7 for detail of vascular pathology causing this condition. (Courtesy Dr. Paul B. MacCready)

Peripheral vascular lesions have been shown to be the causative agent of otosclerosis in five of the six cases presented. Where peripheral vascular pathology will strike is a matter of chance, as evidenced by the types known as Buerger's disease and Raynaud's disease. If peripheral vascular pathology strikes the internal auditory artery, naturally nerve degeneration will result. Also if it strikes anywhere along the auditory neural path to the sylvian gyrus, nerve degeneration would be diagnosed.

EDMUND P. FOWLER, JR., M.D., New York, N.Y.: I should like to ask Dr. Guild some questions. In the first place, what are his thoughts about the paper written by Nager and Fraser in which they showed bone growth inside the lower turns of the cochlea in cases of quite extensive otosclerosis which were actually not otosclerotic growths but a reaction to it in the perilymph. These were the types of changes that you get in a low grade osteomyelitis, something like you get in leukemia, and which Nager and Fraser thought could explain nerve deafness in these cases of very extensive otosclerosis.

FIG. 7.—Detail showing obliterative arteritis. The vessel in the upper right corner shows partial obliteration by hypertrophy of endothelium. Periarterial fibrosis is also present. In the center and lower left corner complete obliteration of small vessels has occurred. Numerous lacunae devoid of bone cells give evidence of necrosis.

I should also like to ask Dr. Guild whether he, too, does not think that one is more likely to get a nerve deafness in the very extensive types of otosclerosis as compared with the rather minor ones, even when Nager-Fraser pathology is not found in the perilymph.

FIG. 8. Otosclerosis of the stapes removed from a man 44 years old during the fenestration operation. Note hypervascularity and enlargement of the posterior crus. Ankylosis of the incudostapedial joint has also occurred. (Lempert Institute, no. 2202)

I should also like to ask him if he has ever seen, and if he does not believe that a focus at the third most common site for otosclerosis, near the end of the first turn of the cochlea, does not occasionally actually invade the modiolus and interfere with the nerve.

Lastly I should like to ask him whether, if the Hopkins Group had tested the hearing at 1000 and 2000 for bone conduction, they might not have found more clinical evidence of nerve deafness, and whether in these cases he might not have found histologic changes if clinical changes were present for those notes which are so much more often involved than 512.

GEORGE E. SHAMBAUGH, JR., M.D., Chicago, Ill.: None of us can dispute Dr. Guild's observations and conclusions. He has simply not seen in the sections at his disposal histologic changes which explained nerve degeneration seen during life in these cases. I think, however, that there are some things which must be explained, and I think it would be a mistake for us to leave tonight with the idea that otosclerosis and nerve degeneration do not occur together very frequently.

The first thing we must explain is why it is that otosclerotics in middle life, when nerve degeneration from other causes is comparatively rare, so often will show nerve degeneration. I would estimate that in our series 30 per cent of the otosclerotics under 40 will have some degree of nerve degeneration as part of their otosclerotic process, as far as we can tell clinically, and certainly nerve degeneration is not that common in the general population. I would estimate under 40 it is perhaps two or three per cent.

Secondly, there is a pattern of nerve degeneration which we see in otosclerosis which is unusual in patients without otosclerosis. There seems to be a bite taken out of the middle of the tone range by this nerve degeneration. The deep notching in the air curve in the middle of the tone range is reflected in the bone curve. This was seen in a young person, aged 25. She had typical bilateral stapes ankylosis. We did a fenestration operation and were able to get a good improvement, but she still has a notch or bite out of the middle of her tone range due to this nerve degeneration.

Nine years after operation the bite has increased. The high tones remain the same. The low tones remain the same. In other words, this patient had a progressive deterioration of her nerve function localized to a very small area in the cochlea. This is a most unusual thing. I have never seen it in a patient without otosclerosis and it has occurred in several patients with otosclerosis, so we think that this is one of the characteristic, although unusual, patterns of nerve degeneration associated with otosclerotic stapes fixation.

I think it is up to us to try to get histologic studies on cases like this after death in order to discover just what the pathologic condition is. I am sure that pathologic changes are there which are due to the otosclerosis, and the fact that Dr. Guild has not seen it in his small series does not rule it out.

BENJAMIN SHUSTER, M.D., Philadelphia, Pa.: I should like to have Dr. Wolff clear up one point for me. I assume from the slides that otosclerosis might be a vascular disease. All cases of otosclerosis do not show these vascular changes. But the point I should like to make is that if it is a vascular disease, and she assumes that this vascular disease extends into the inner membranous labyrinth, how can we correlate the fact that the

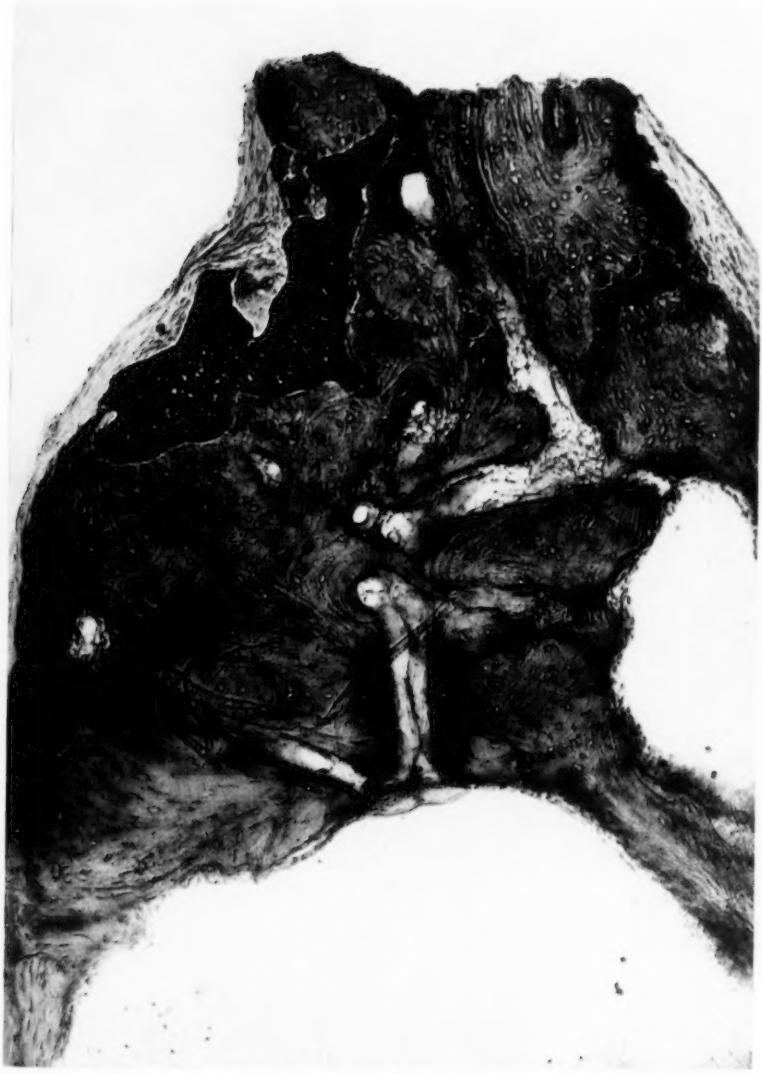

FIG. 9.—Detail showing ankylosis of malleoincudal joint. Only a fragment of cartilage remains beneath the joint ligament. The bone has been hypervascular but the vessels are now atrophic. (Lempert Institute, no. 2202)

membranous labyrinth gets its blood supply from an entirely different source than that supplying the middle ear and the bony capsule and there is absolutely no connection at all?

I believe that Dr. Shambaugh, Sr., worked on that problem and was never able to determine any collateral circulation between the middle ear or mesodermic area and that of the membranous inner ear. If she finds le-

sions in both areas, they cannot be those of otosclerosis but indicate some vascular disease with its effect on the inner ear structure.

VICTOR GOODHILL, M.D., Los Angeles, Calif.: Dr. Guild has shown in his histologic studies that round window lesions do occur in otosclerosis. The clinical picture painted by Dr. Shambaugh has been seen by many of us. There appears to be more so-called neural degeneration in the younger than the older otosclerotic patients.

May it not be that in the fulminating young otosclerotic we frequently encounter simultaneous fixation of both oval and round windows, a situation which conceivably may produce phase relationship changes which may produce the audiometric picture of so-called neural deafness?

DR. GUILD: I wish first to thank each and every one who has taken part in this discussion. I am sorry that it is going to be impossible to answer some of the questions. I have the feeling that my opinion is not in accord with that of the group.

Before starting on the specific items I should like to express the hope that if the paper, plus what has been said, has done nothing more than help us to be careful in how we describe a condition, it has been worth while.

Dr. Wolff, I could spend hours discussing those sections. I delight to see them. But I don't feel that we should take the time of the audience here.

Dr. Shuster's comments about Dr. Wolff's discussion took the words out of my mouth. As he said, the studies of Dr. Shambaugh, Sr., of Eichler and of the others who have investigated the problem, showed that there is no communication between the two systems of blood supply. But if we are dealing, as Dr. Wolff is suggesting, with a systemic tie-up with the adrenal cortex, there is, of course, another possibility. The thing, Dr. Wolff, that I did not see in your sections was the nerve degeneration itself. I was watching for those sections of the cochlea, and I hope that sometime you will write a paper on the subject showing what was there with respect to the condition of the cochlear nerve and end organ.

Dr. Fowler, you asked four heavy questions and the hour is late. I know the Nager-Fraser paper. I have known it for many years. Our collection does not contain an example of the condition they described. I have asked various persons who have done many fenestrations whether they have ever had the impression at operation that they were dealing with an otosclerotic area as great and extensive as that of the Nager and Fraser case. I have not had any positive answers yet. I don't know that I have asked you that question but I might do it. The suggestion is, of course, that we are dealing essentially with the pressure of a tumor in such cases rather than with the toxic action that most of us are thinking of when we speak of nerve degeneration secondary to otosclerosis. That would apply also to cases with a focus at the anterior part of the first turn. Dr. Wolff's suggestion is much more to the point, namely, a systemic toxic effect for some reason.

If we had tested at 1000 and 2000 is a hypothetical question. We did not test bone conduction at these frequencies. I might comment, however, that these tests, had they been made, would not have altered the histopathologic findings, either for the cases with otosclerotic ankylosis or for those without. My conclusions would therefore have remained unchanged.

Dr. Meltzer, I have seen so-called malignant otosclerosis clinically but not histologically, so I know exactly what you mean. Such cases have not come to autopsy, and those for whom we have records will probably outlive me. That is the sad part about getting the answer there.

Dr. Shambaugh, what I said in general bears particularly on your remarks. As to those notches and the high incidence of what you term "nerve degeneration," I would much prefer at the present state of our knowledge to stick to the description of the hearing loss and merely say that it can be caused by a nerve lesion but it may be due to something in the physics of sound conduction that we do not know about. Our collection of histologic otosclerosis is small compared with your clinical series and with many of the others. But it is the largest histologic collection extant, and it certainly does not show a 30 per cent incidence of "nerve degeneration."

The same thing applies, Dr. Goodhill, to your suggestion about the round window and the possible phase relation. I would much prefer to think that we are dealing with something in the physical system of sound transmission than with "nerve degeneration."

LESIONS OF THE ORGAN OF CORTI

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THE STUDY of cochlear pathology in animals with experimentally produced deafness has provided sufficient new information to justify an attempt to correlate it with some of the psychoacoustic phenomena associated with deafness.

Some of the correlations are adequately supported. For others there is only circumstantial evidence.

The evidence for the conclusions drawn in this paper is derived mainly from the findings in a series of 33 experimentally deafened animals. These were chronic experiments. Sterile technic was used in the operative procedures. The binocular microscope was used as needed. Anesthesia was accomplished with nembutal injected intraperitoneally. Penicillin was administered postoperatively. The following methods were used to produce the lesions: (1) needle punctures of the cochlea, (2) electrocautery after thinning the cochlear wall, (3) blows to the head, and (4) partial section of the cochlear nerve. Care was taken not to injure the sound conducting mechanism of the ear. Audiograms were obtained by the behavioral method which is probably reliable within a range of plus or minus 5 decibels. The method is described in detail elsewhere.[15] The cochleas were graphically reconstructed using the method of Guild.[7] Reconstructions were made for both the organ of Corti and spiral ganglion. These reconstructions were superimposed upon each other so as to allow a correlative study of anatomically related elements within these structures.*

THE FIELD OF EXCITATION

From these studies some observations can be made on the localization and spread of excitation within the cochlea for pure tone auditory stimuli. The audiograms and cochlear charts for four representative ears appear in figure 1. In cat 1 a 3 mm. lesion was produced in the apex of the cochlea by a needle puncture. The greatest hearing loss was for the low frequencies, 22 db. for 125 c.p.s. and 28 db. for 250 c.p.s. The evidence suggests that at near threshold intensities the low frequencies critically excite only those receptors located near the apex of the cochlea.[17]

The injury in Cat 2 is the result of high frequency electrical current applied to the cochlear wall near the apex in an area previously thinned with a dental bur. The sensory and neural elements are degenerated in the apical 6 mm. of the cochlea. The threshold for low frequencies is depressed about 35 db. Thus, at an intensity 35 db. above normal threshold the very low frequencies are apparently capable of exciting

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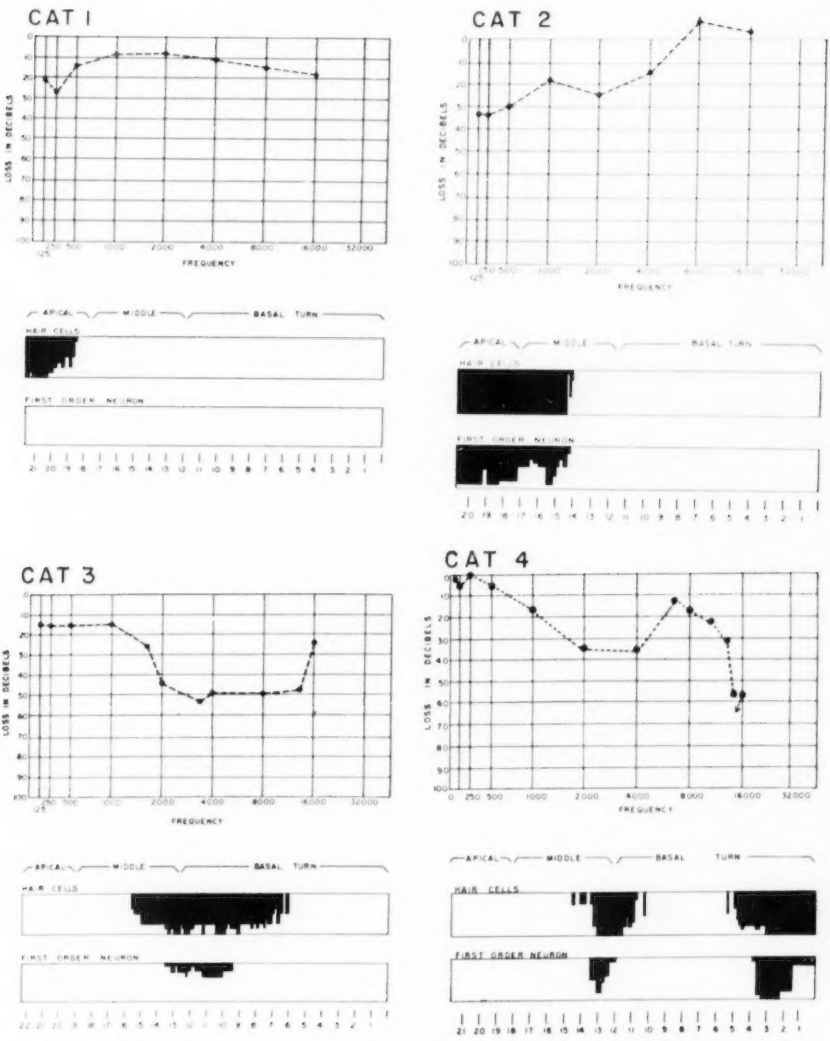
* The reader should be aware when examining the cochlear charts in the illustrations that the black filling represents normal elements in some charts and abnormal elements in others. This inconsistency has occurred because the graphs were originally prepared for several different publications. On the illustrations and in the legends it is clearly indicated whether black represents normal or abnormal structures.



BLACK ABNORMAL

FIG. 1.—The behavioral audiograms and cochlear charts for four animals after experimental lesions in the cochlea. The audiograms indicate the difference in decibels between the preoperative and postoperative thresholds. The frequencies are arranged on the anatomical frequency scale, thus the frequencies above 1000 c.p.s. are plotted on the logarithmic scale whereas those below 1000 c.p.s. are plotted arithmetically so that each 100 cycle interval is equal to the distance between 900 and 1000 on the logarithmic scale. The cochlear charts are placed beneath the audiograms with the basal end to the right. The numbers indicate the distance in millimeters from the basal end. The amount of black filling indicates the percentage of elements degenerated.

Cat 1. Needle lesion in apex. Sacrificed after three months. Cat 2. High frequency electrocautery applied to wall of cochlea near apex. Sacrificed after three months. Cat 3. Experimental head blow. Sacrificed after one month. Cat 4. Needle introduced deeply into cochlea through the round window membrane. Sacrificed after six weeks.

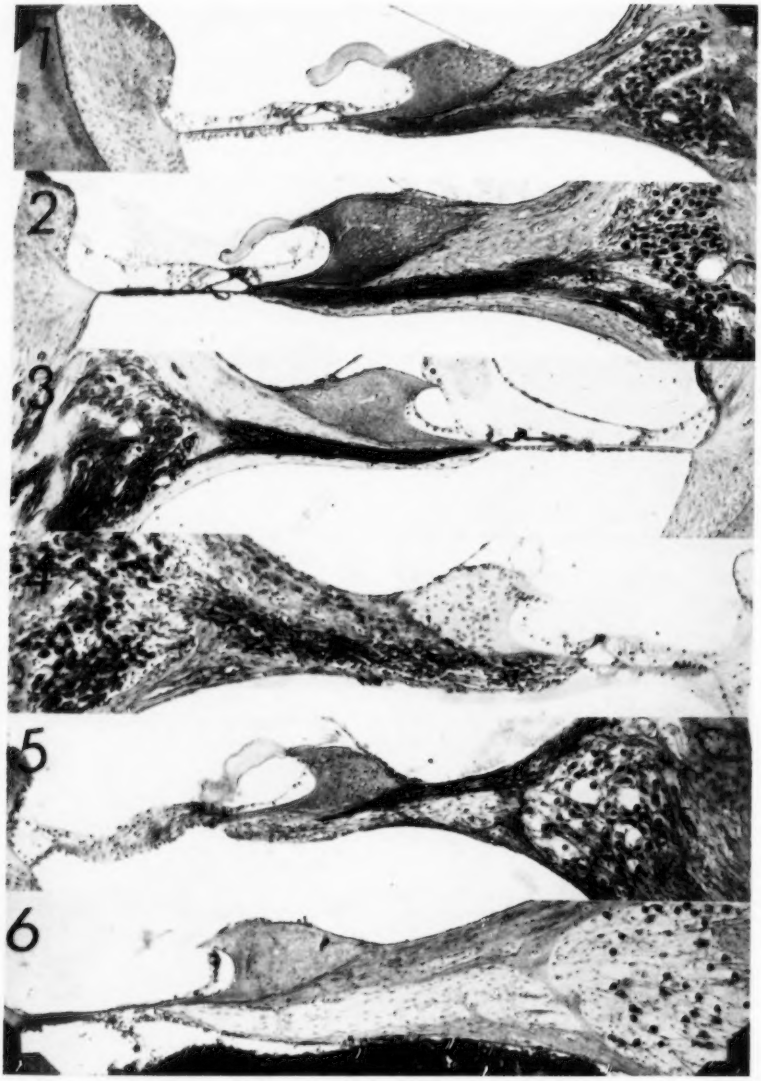


FIG. 2.—Sections of the organs of Corti and spiral ganglion from six ears demonstrate the effect of hair cell loss on the auditory threshold. 1. Normal organ of Corti and spiral ganglion with normal auditory threshold. 2. From the 8 mm. area of the cochlea of Cat 3 (fig. 1). There is a loss of outer hair cells and a 50 db. elevation in auditory threshold for frequencies acting upon this area. The spiral ganglion is normal. The injury was created by a head blow. 3. From the 12.5 mm. area of the cochlea of Cat 9 (fig. 7a). There is a total loss of outer hair cells and a partial loss of inner hair cells. The auditory threshold was elevated about 70 db. for frequencies acting upon this area. The spiral ganglion is normal. The injury was caused by a needle introduced into the apex. 4. From the 7 mm. area of the cochlea. The injury was created by introducing a needle through the round window membrane into the cochlear duct of the basal coil in the 5.5 mm. area.

receptors located 6 mm. or more from the apex. Presumably this area is four or five millimeters from the point of maximum excitation for those frequencies.

The cochlear injury in Cat 3 was produced by delivering a blow to the head.[16] The lesion consists of a loss of outer hair cells from the 6 to 15.5 mm. points. There was a slight decrease in the number of ganglion cells supplying the cochlea in this region. The audiogram for the ear showed a 50 db. depression for the frequencies from 2000 c.p.s. to 12,000 c.p.s.

The lesion in the ear of Cat 4 was created by introducing a needle deeply into the cochlea through the round window membrane so that a lesion was created in both the basal and middle coils.[19] There are two depressions in the audiogram corresponding to the two lesions in the cochlea. The abrupt hearing loss for high frequencies corresponding to the basal end lesion suggests that the field of excitation for these frequencies does not spread greatly in an apical direction with increasing intensity. Correlation of the audiogram with the second cochlear lesion indicates that the field of excitation for the frequencies 2000 c.p.s. to 4000 c.p.s. does not spread beyond an area of about 4 mm. for intensities up to 35 db. above normal threshold.

The results in all animals have shown without exception that the auditory spectrum has a spatial representation within the cochlea, low frequencies in the apex, high frequencies in the base. At near threshold intensities the field of excitation is relatively small. As intensity is increased there is a rapid growth of the field of excitation for low frequencies, less so for high frequencies.

DEGREES OF HAIR CELL INJURY AS RELATED TO AUDITORY THRESHOLD

Many of the experimental ears have lesions in the organ of Corti extending over an area of several millimeters in which there is a rather uniform loss of hair cells. Only in such ears are correlation studies of hair cell loss and auditory function meaningful (fig. 1, Cat 3). For small lesions it is not possible to determine with certainty whether threshold responses are due to excitation of receptors existing within the lesion or to uninjured elements at the periphery of the lesion. Sections of the organ of Corti and spiral ganglion have been selected from six ears to show the effect of progressive stages of degeneration of the end organ on the auditory thresholds (fig. 2). A partial loss of outer hair cells seems to result in a hearing loss for the involved frequencies of less than 50 db. In the entire series of ears several have areas with total loss of outer hair cells. They consistently show a hearing loss of about 50 db. for the frequencies involved. The absence of some inner hair cells in addition to a total loss of outer hair cells creates hearing losses greater than 50 db. These relationships apply to lesions involving frequencies up to 4000 c.p.s., whereas for higher frequencies the hearing losses are correspondingly more severe. A complete loss of hair cells throughout

(Legend for figure 2, continued)

All hair cells are missing. The animal made no response when frequencies acting upon this area (10,000 to 12,000 c.p.s.) were delivered at maximum intensity available (80 db. above normal threshold). The spiral ganglion is normal. 5. From the 16.7 mm. area of the cochlea of Cat 2 (fig. 1). The organ of Corti is flattened. All hair cells are missing. There is about 50 per cent loss of spiral ganglion cells. The auditory threshold was elevated about 20 db. for frequencies acting upon this area (about 1000 c.p.s.). Presumably at this intensity normal elements were being stimulated in the 14 mm. area in this ear (fig. 1, Cat 2). The lesion was created by electrocautery to the cochlear wall near the apex. 6. From the 7.5 mm. area of the cochlea of a cat which received a blow to the head. Following the injury the animal made no responses to auditory stimuli at the maximum intensities available (60 to 90 db. above normal threshold). The organ of Corti is completely missing and the spiral ganglion is severely degenerated. No peripheral nerve fibers can be seen.

the cochlea renders the animal totally deaf.

A loss of all the hair cells in a region of the basal coil causes a total deafness for the affected frequencies. On the other hand, a total loss of hair cells in the apical region only does not abolish the auditory response to the low frequencies. It can, therefore, be inferred that at high intensities the field of excitation for low frequencies spreads to involve receptors located at a considerable distance towards the basal end. Probably in such an ear, pitch would be displaced upward for the low frequencies. In none of the animals is there evidence that hearing can occur in the absence of hair cells. In other words, the nerve endings of the spiral ganglion cells cannot be excited directly by sound stimuli. The evidence suggests that the intensity required to stimulate outer hair cells is lower than for inner hair cells.

EFFECT OF END ORGAN INJURY ON THE SPIRAL GANGLION

In experimentally produced cochlear lesions it is common to find that the decrease in hair cell population greatly exceeds the loss of spiral ganglion cells. It is actually possible for all the hair cells in a region of the cochlea to disappear while the spiral ganglion in this area remains normal.

In experimentally produced cochlear lesions, degenerative changes in the spiral ganglion appear to parallel more closely the extent of injury suffered by the supporting cells of the organ of Corti, particularly the pillar cells. The organ of Corti may be considerably flattened and devoid of hair cells, but so long as the pillars are erect there often is very little or no loss of spiral ganglion cells. There are exceptions to this rule. There is a type of degenerative change which is characterized by the presence of a homogeneous eosinophilic deposit in the fluid spaces of the organ of Corti, particularly Corti's tunnel. In the presence of this process there may be severe degenerative changes in the spiral ganglion even though the pillars appear normal. When there is a shortening of the height of the pillar arch, or a partial collapse of the inner or outer pillars in a region of the cochlea, there is usually some loss in the number of ganglion cells in that area. A total loss of pillar cells usually is associated with a severe loss of spiral ganglion cells. Representative sections demonstrate the effect of degenerative changes in the organ of Corti on the spiral ganglion (fig. 2). This relationship is obvious in the ear of an animal which had sustained a severe hearing loss and a widespread cochlear lesion as the result of an experimental head blow. The condition of the hair cells, pillar cells, spiral ganglion and auditory acuity in this ear are plotted together in the accompanying graph (fig. 3). The graph clearly demonstrates that the pathology in the spiral ganglion more closely parallels pillar cell injury than hair cell loss. There are exceptions to this rule; for example, the cochleas of congenitally deaf Dalmatian dogs may have a normal spiral ganglion even though the organ of Corti is flattened and the pillars are severely degenerated.*

It is a common occurrence in experimental injuries to the cochlea to find a few ganglion cells remaining in a region of the cochlea in which the organ of Corti is entirely missing. These ganglion cells appear to have survived a sublethal injury and persist in a pathological state. The nucleus is somewhat pyknotic and placed toward the axon side. The peripheral fiber (dendrite) is missing and

* From the histologic collection of W. D. Neff, Laboratory of Physiological Psychology, University of Chicago.

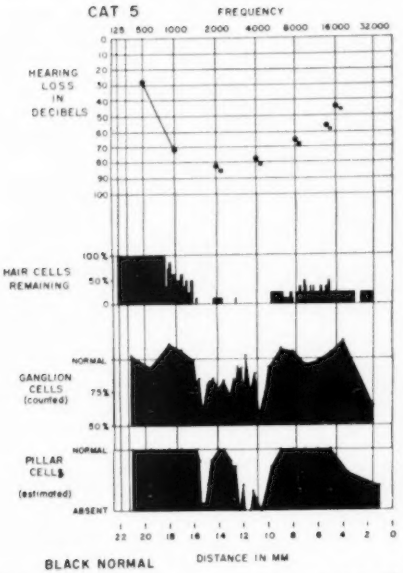


FIG. 3—Animal suffering a severe hearing loss and a diffuse cochlear lesion following a blow to the head. The black filling represents per cent of normal elements remaining. The data for the spiral ganglion is based on counts of ganglion cell density in Rosenthal's canal. The criteria for pillar cell injury was arbitrarily based on a gradient of ten, with ten representing normal pillars and zero representing a total loss of the pillars. The stimulus frequency is indicated at the top and the distance along the organ of Corti at the bottom of the chart. The change in spiral ganglion correlates better with the pillar cell injury than the hair cell loss.

a large vacuole is present at the dendritic pole of the cell.

THE EFFECT OF INJURY TO THE SPIRAL GANGLION

The cells of the spiral ganglion degenerate following direct mechanical injury to either their central or peripheral fibers. Regeneration does not occur. Although the spiral ganglion may degenerate completely, the organ of Corti remains histologically normal. For studies on section of the axon see the work of Wever and Neff.[21] For an example of selective mechanical injury to the dendrite without damage to the organ of Corti see the report by Hind and Schuknecht.[11] The findings in two animals indicate that auditory thresholds for pure tones may be normal in spite of over 50 per cent reduction in ganglion cells in the region of the cochlea serving those frequencies.[20] Microphotographs from three animal ears show degenerative changes in the spiral ganglia and normal organs of Corti following experimental section of the cochlear nerve (fig. 4).

DIPLACUSIS

The audiogram and cochlear reconstruction for the ear of a cat which was injured by a blow to the head are shown in figure 5a. The type of injury to the cochlea caused by a blow to the head differs in no way from the lesion caused by intense air conducted sounds.[18] There is degeneration of outer hair cells in a 3 mm. region of the upper basal coil and a hearing loss of 45 db. for 4000 c.p.s. and 38 db. for 8000 c.p.s. There is a loss of about 20 per cent of the ganglion cells supplying the 9.4 to 10.1 mm. area (fig. 5b). The approximate location of the areas of maximum excitation for the 8000 c.p.s. and 4000 c.p.s. frequencies are indicated on the chart (fig. 5 b and c). These points were determined from correlative studies on a number of ears having lesions of the basal coil with high tone hearing losses. The graph demonstrates a great disproportion between sensory and neural elements in the area of injury as a result of the severe loss of outer hair cells. The small dots represent hair cells with grossly abnormal morphology and it is doubtful that they are capable of normal function. In the ear of Cat 6 it would seem entirely possible for the frequency 8000 c.p.s. at the threshold intensity of 38 db. to be stimulating outer hair cells in the 8.1 mm. area rather than inner hair cells in the 8.4 mm. area. Two facts support this prob-

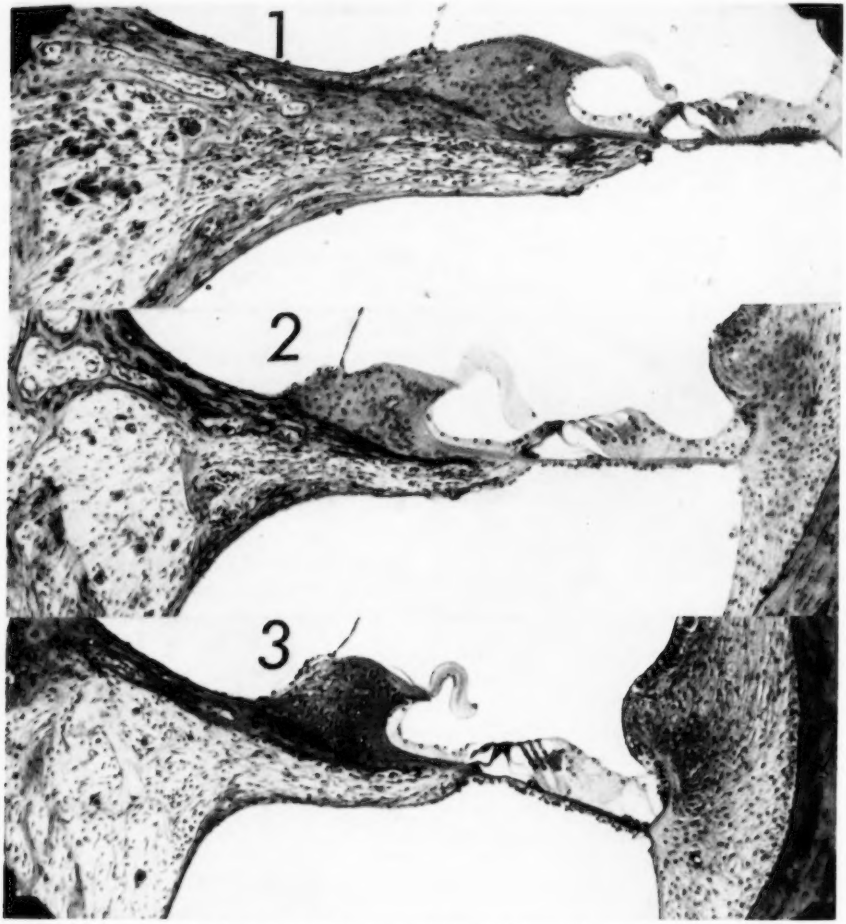


FIG. 4—These sections are from ears with experimental lesions of the cochlear nerve. 1. From the 7.7 mm. area. About 35 per cent of the ganglion cells remain as based on actual counts of ganglion cell density. The auditory threshold was normal for the frequency activating this area (8000 c.p.s.). The organ of Corti is normal. 2. From the 15.8 mm. area of the same ear. About 10 per cent of the ganglion cells remain (counted). The threshold for the 1000 c.p.s. frequency, which is thought to act upon this area, was 55 db. However, this frequency may have been exciting more normal receptors located in the basal direction. The organ of Corti is normal. Sacrificed five weeks after injury. 3. From the 7 mm. area. The spiral ganglion is completely degenerated. The organ of Corti is normal. There was no response to frequencies higher than 1000 c.p.s. delivered at high intensities. Sacrificed four months after injury.

ability: (1) A stimulus intensity much greater than 50 db. above normal threshold is ordinarily required to produce a response in an animal having only inner hair cells remaining in this region of the cochlea (fig. 7a). (2) It is known that with increasing intensity the spread of excitation is mainly in a basal direction. If it is true in this injured ear that the 8000 c.p.s. stimulus at an intensity of 38 db. above normal threshold has its main effect on receptors in the 8.1

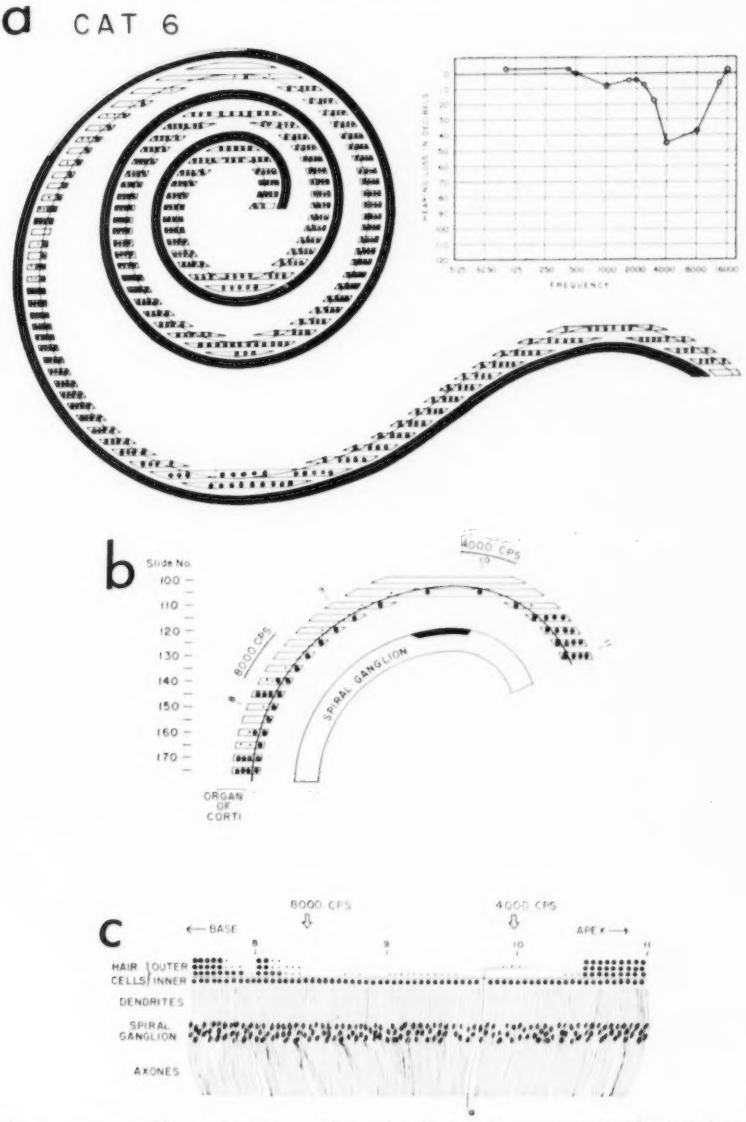


FIG. 3. a. Cat 6. The graphic reconstruction and audiogram for an animal which received an experimental blow to the head. The animal was sacrificed three weeks after the injury. The quadrilateral boxes represent organs of Corti and the dots represent hair cells. A small dot is an abnormal hair cell. The accompanying spiral channel represents the spiral ganglion, and the black filling the percentage of cells remaining.

b. Another graphic portrayal of the lesion in the upper basal coil. The distance in millimeters from the basal end is shown as well as the areas of maximum importance for the frequencies 8000 c.p.s. and 4000 c.p.s. The ganglion lesion is represented in black.

c. A third diagrammatic representation of the pathological anatomy of the sensory and neural elements in this ear. The diffuse loss of outer hair cells is shown. There is only a slight loss of spiral ganglion cells. Theoretically, diplacusis and recruitment should exist in such an ear.

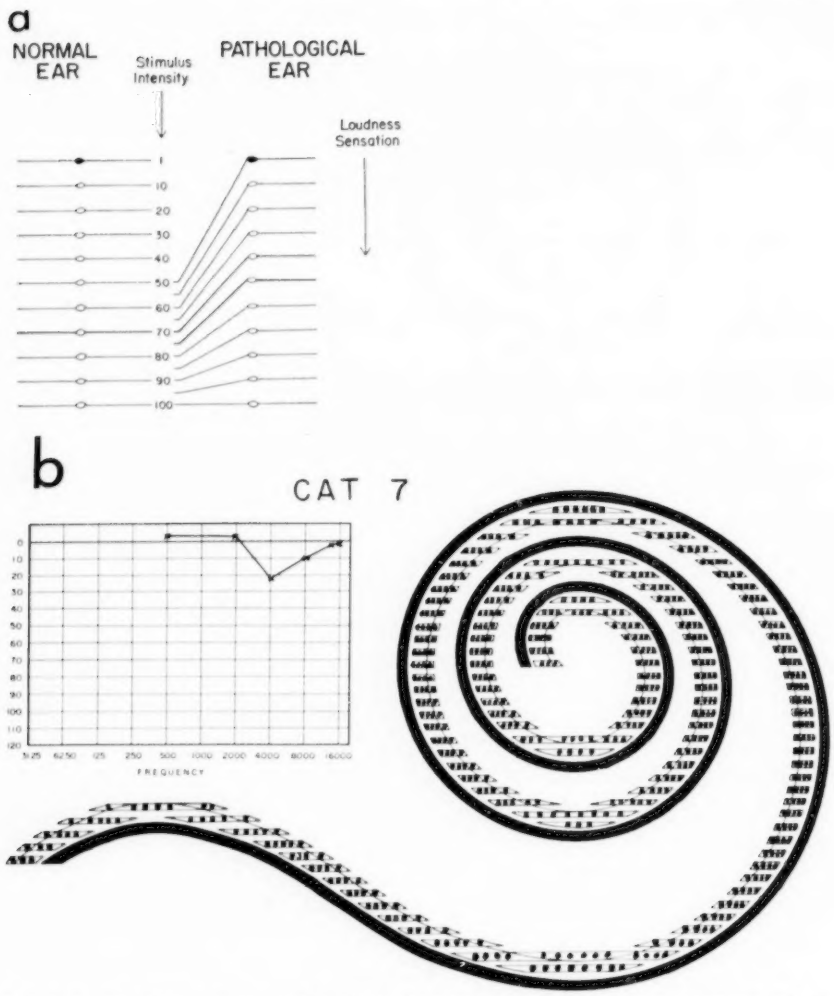


FIG. 6—a. This diagram is presented to clarify the relationship between loudness recruitment and the decreased intensity difference limen.

b. The audiogram and graphic reconstruction for an animal which received an experimental head blow. The animal was sacrificed six weeks after the injury. The cochlea appeared normal even though there was a threshold elevation of 20 db. for the 4000 c.p.s. frequency.

mm. area rather than the 8.4 mm. area, then theoretically the perceived pitch would be higher (diplacusis).

If we also assume that at very high stimulus intensities the inner hair cells in the 8.4 mm. area are maximally excited so that they activate large numbers of nerve fibers, then the point of maximum neural activity might well return to the 8.4 mm. area as in the normal ear and diplacusis would disappear.

Davis and co-workers[2] performed an exhaustive study of stimulation deafness in humans and made the following observations: (1) a hearing loss restricted to a narrow range of frequencies may cause severe distortion of pitch perception, (2) large displacements of pitch are always upward, and (3) displacement of pitch diminishes as the intensity of the test tone is increased.

LOUDNESS RECRUITMENT

The physiological correlate of loudness is thought to be the total number of impulses per second occurring in all fibers, the variables being the number of nerve fibers involved and the frequency of the impulses in these fibers. Thus in the ear of Cat 6 (fig. 5) if it were possible at high intensities for either the 8000 c.p.s. or 4000 c.p.s. frequencies, acting upon the inner hair cells, to excite a normal complement of nerve fibers, there would be normal loudness sensation at these high intensities (recruitment).

The recruitment phenomenon was first described by Fowler[6] as occurring in patients with perception deafness. Simply stated it means that with increase in stimulus intensity there is an abnormally rapid rise in loudness sensation, so that even though the threshold is elevated, loudness sensation at high stimulus intensities will approximate or equal that of a normal ear. Dix, Hallpike, and Hood[4] were the first to note the absence of this phenomenon in patients with primary lesions of the cochlear nerve. Because of its presence in Ménière's disease and stimulation deafness and absence in cochlear nerve lesions, the recruitment phenomenon is now considered to be a manifestation of hair cell injury. Lüscher[14] has shown that an ear having loudness recruitment also is better able to detect small changes in stimulus intensity (decreased intensity difference limen). Hood[12] has demonstrated subsequently that loudness recruitment is a transient phenomenon lasting but a short time after the onset of the tone. He has suggested that recruitment is a manifestation of the on-effect and has applied the term adaptation to this rapid decay in loudness. The on-effect is stated to be an initial high frequency discharge in the action potential developed from stimulation of an end organ.

The relationship between loudness recruitment and decreased intensity difference limen is diagrammatically shown in figure 6a. On either side of a decibel intensity scale are a series of schematically drawn bipolar cells each of which represents a unit number of spiral ganglion cells. The left side of the graph applies to the normal ear. For this ear it is shown that at 1 db. of intensity the first neural unit is excited, at 10 db. two units, at 20 db. three units, et cetera, and that loudness sensation increases proportionately. The right side of the graph shows the condition as pertains to the abnormal ear with the pathological anatomy required for loudness recruitment. Because of an injured organ of Corti the first neural unit is not excited until an intensity of 50 db. is reached. However, as intensity is increased, neural excitation proceeds at an abnormally rapid rate so that at 100 db. the entire complement of neurons is excited and loudness sensation is as great as in the normal ear. It is also clear that unit changes in intensity (occurring within the sensitivity ranges of these ears) would affect more fibers and hence create greater changes in loudness sensation in the pathological ear that in the normal ear, the psychoacoustic correlate being a decreased intensity difference limen.

The means by which residual inner hair cells in a pathological ear can activate large numbers of nerve fibers at high stimulus intensities is not entirely clear. It is known that a great number of

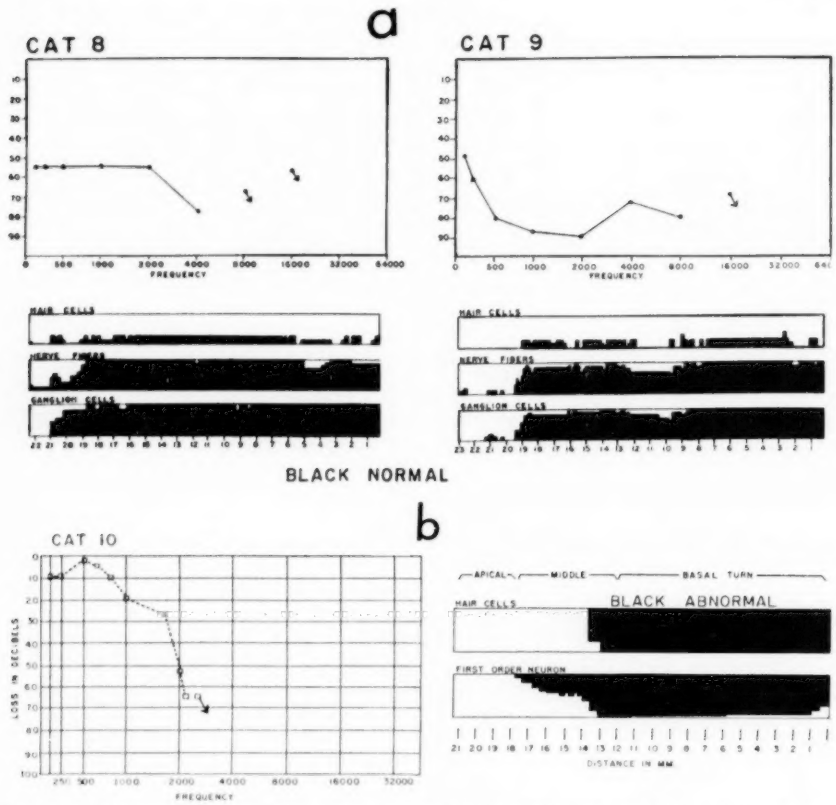


FIG. 7—a. The audiograms and cochlear charts for two ears injured by the introduction of a needle into the apex. In these charts the height of the black filling indicates the estimated percentage of normal elements remaining. For each ear the hearing loss is greatest for the high frequencies even though the lesion is most severe in the apex.

b. The cortical response curve and cochlear charts for an animal which was found to exhibit an abnormal threshold during an electrophysiological investigation of the auditory cortex. The degenerative changes in the sensory and neural elements are almost equal. Black indicates percentage of elements missing.

the peripheral nerve fibers have their endings only on external hair cells. (See the recent work of Fernandez.[5]) This is demonstrated by fiber "a" in figure 5c. One explanation may be that an electrical excitatory field develops around the inner hair cells as a result of stimulation and that it becomes sufficiently large and strong at high stimulus intensities to activate nerve fibers lying in the vicinity of the cell. These additional fibers apparently are activated for only a short time during the initial intense discharge known as the on-effect. As the potential diminishes, the hair cells are capable of activating only those fibers having a more intimate relationship with them, so the loudness sensation diminishes.

Therefore, loudness recruitment and decreased intensity difference limen appear to be manifestations of an incomplete sensory lesion (with a normal or nearly normal spiral ganglion).

It would be possible theoretically to have an elevation of the auditory threshold as the result of a sensory lesion and not have recruitment under the following conditions:

1. A loss of all the hair cells in a relatively small section of the organ of Corti. In such an ear the affected frequencies presumably would activate the more normal elements at the borders of the lesion. Diplacusis should be present and should not be lost with increase in stimulus intensity.

2. An incomplete sensory lesion of any size associated with a partial loss of ganglion cells.

In clinical experience it is not unusual to find recruitment for some frequencies and not for others in the same ear. It must be assumed that the pathological anatomy in such an ear is not uniform and that the sensory-neural relationships are right for recruitment in some areas and not in others. The variations in the quantity of sensory cells and ganglion cells in various parts of the cochlea of Cat 5 (fig. 3) suggest that this assumption is not entirely unreasonable.

MÉNIÈRE'S DISEASE

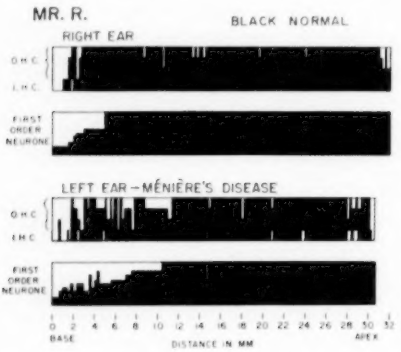
The cause for the deafness in Ménière's disease has been a topic for much speculation. The most popular conceptions are: (1) that the endolymphatic hydrops in some manner prevents the physical stimulus from being impressed upon the vibrating structures of the inner ear, and (2) that the disease creates a degenerative change in the end organ, particularly the hair cells.

Early in the course of Ménière's disease it is characteristic for the hearing loss to be most severe for the low frequencies. As the disease progresses it is common for the entire range of frequencies to be involved and in many cases the hearing loss eventually is more severe for the high frequencies. Experimental studies have shown that a low tone perception type hearing loss will result from a degenerative change in the organ of Corti or spiral ganglion in the apical region of the cochlea. This suggests that the low frequency hearing loss occurring early in the course of Ménière's disease is due to involvement of the apex of the cochlea. It may be that the apical region receives the most severe abuse throughout the course of the disease even though the hearing loss may ultimately become more severe for high frequencies. Support for the latter statement is presented in figure 7a, which shows the audiograms and cochlear charts for two cochleas in each of which a needle was introduced into the apex. The lesions are diffuse; however, the most severe degenerative change is seen in the apical coil. In both ears it appears that even though the loss in hair cell population is greater in the apical region than in the basal region, the hearing loss for low frequencies is less severe than for high ones.

The histologic findings in human ears with Ménière's disease usually have revealed the spiral ganglion to be not significantly different from those of ears without Ménière's disease. The fact that loudness recruitment is marked in Ménière's disease would lead one to suspect that the deafness is caused by hair cell injury. However, changes in the organ of Corti and its hair cells have not been remarkable in the ears we have examined. Small changes, of course, would be masked by postmortem degeneration.

Dix and Hallpike[3] have described changes in the staining characteristics and structure of the organ of Corti in Ménière's disease. The disease may have been more advanced in their cases. The charts appearing in figure 8a were made from cochlear reconstructions and illustrate the findings in the sensory and

neural structures in both ears of a patient having Ménière's disease in the left ear. The history and histologic findings have been reported by Lindsay.[13] In 1942 this 47 year old patient fell during an attack of vertigo, suffered a fracture of the skull and died of a subdural hematoma. The temporal bones were acquired from Dr. Lawrence R. Boies of the University of Minnesota. Three years before death the patient had been examined at the Mayo Clinic and was believed to have Ménière's disease. At that time the hearing was normal in the right ear for frequencies 2048 c.p.s. and below. An abrupt threshold loss was noted for frequencies above 2048 and a marked loss for all higher frequencies. In the left ear there was a 30 db. loss for all frequencies tested up to 2048 c.p.s. and an abrupt loss for higher frequencies. Histologic studies showed a marked dilatation of the endolymphatic spaces in the left ear. Both ears have a degenerative change at the basal end, which is not an unusual finding in persons of this age. The size, shape, and staining characteristics of the organ of Corti are the same in both ears. Postmortem degenerative changes are moderately severe and equal in both ears. The hair cells were all in a stage of degeneration but could be identified and charted (fig. 8b). A few more hair cells were missing in the left ear than in the right but the difference is not striking. It was known that at least a 30 db. hear-



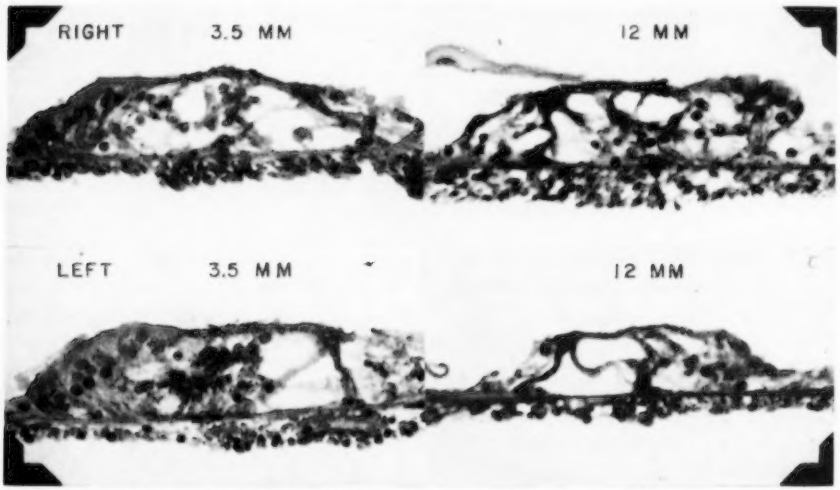


FIG. 8.—a. Cochlear charts for a patient with Ménière's disease in his left ear. The height of the black filling indicates the estimated percentage of normal elements remaining.

b. The top two photomicrographs were taken from the 3.5 mm. and 12 mm. areas of the right ear. The lower two came from the 3.5 and 12 mm. areas of the left ear with Ménière's disease. The general appearance of the organ of Corti is similar in the two ears.

ing loss was present for all frequencies in the diseased ear three years prior to death. This deafness apparently had occurred without a great loss in hair cell population. Nevertheless, it is quite possible that a change in the hair cells is the cause for deafness in Ménière's disease. It is possible, for example, to produce mild "permanent" hearing losses in animals without creating histologically demonstrable changes in the supporting structures or hair cells of the organ of Corti. The audiogram and cochlear reconstruction for an animal receiving a blow to the head are shown in figure 6b. Twenty-four hours after the injury there was a moderately severe hearing loss involving the entire frequency range but most severe for the 4000 c.p.s. frequency. There was an average improvement in the threshold of 19 db. during the time interval between the twenty-fourth and forty-eighth hour after the injury. The audiogram remained stable for the next six weeks at the level shown, at which time the animal was sacrificed. No changes could be found in the cochlea to explain the hearing loss.

Thus the accumulated evidence indicates that although there is a widespread disturbance of physiology in the cochlea in Ménière's disease, the dysfunction is most severe in the apical portion. Furthermore, the consistent presence of loudness recruitment in Ménière's disease strongly supports the contention that the function of the hair cells is affected. In the early stages of the disease this effect apparently is sublethal to the cell because there is very little decrease in hair cell population. However, more human ears in a better state of preservation must be studied before these points are conclusively proven. Until this is done, the possibility of an interference with conduction of the physical stimulus through the cochlea remains a possibility.

HIGH TONE DEAFNESS

The type of perception deafness which begins at the high end of the frequency scale and progresses slowly to involve lower frequencies over a period of years has as its underlying pathology a degenerative change in the basal coil usually involving both the organ of Corti and the spiral ganglion. For a classical study on the pathology of high tone deafness see the report by Crowe, Guild and Polvogt.[1]

This lesion is thought to be related to the aging processes. Guild has pointed out, on the other hand, that an insidious, slowly progressive high frequency hearing loss may begin in childhood.[9]

The degenerative changes usually involve the hair cells and spiral ganglion rather uniformly both as to severity and location. Any disparity, when it exists, usually consists of greater degeneration in the spiral ganglion than the organ of Corti. Thus the pathological anatomy in high tone deafness is not of the type which is thought to be necessary for loudness recruitment. A cortical response curve and cochlear charts for an animal which was found to exhibit an abnormal threshold during an electrophysiological investigation of the auditory cortex are shown in figure 7b. The points on the curve represent deviations from normal of the stimulus intensities required to elicit cortical strychnine spikes in response to the onset of tone pulses.[10,11] The degenerative change in hair cells is severe throughout the basal 13.75 mm. of the cochlea, at which point there is a rather abrupt return to normal. The degenerative change in the spiral ganglion is severe to 13 mm. and improves progressively to become normal at 17.8 mm. There are also three examples of pre-existing high tone deafness in the series of 33 experimental animals on whom behavioral tests of hearing were made. The pathology

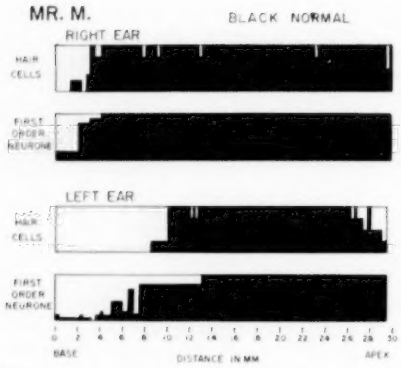


FIG. 9a—Cochlear charts from the right and left ears of a 47 year old patient with bilateral histologic otosclerosis. The black filling indicates the estimated percentage of normal elements remaining. The lesions in the basal coil are not more severe than the lesions found in ears without otosclerosis.

parallels that described by Guild for humans.

A considerable number of patients having clinical otosclerosis have a mixed type of deafness. The perceptive loss involves primarily high frequencies. It is usually slowly progressive, as is the high tone deafness found in ears without clinical otosclerosis.[8] In a study of 81 ears of patients with histologic otosclerosis Guild found that atrophy of the cochlear nerve fibers or the organ of Corti was no more frequent than in ears free from otosclerosis. There was stapes ankylosis in only ten of these ears. The findings for a patient with severe histologic otosclerosis without stapes ankylosis appear in figure 9a. In both ears there were large otosclerotic foci along the margins of both the oval and round windows. In both ears there was bulging of the oval window lesion into the scala tympani of the basal coil, more marked on the left. The charts show a degenerative change in the organ of Corti and spiral ganglion in the basal coil of both ears, more extensive in the left. It is not known whether these sensory-neural lesions are the result of the otosclerotic lesions. Similar findings occur in ears without otosclerosis. The charts do show the pathologic anatomy existing for a basal end lesion in the human. As in the cat ears with pre-existing basal lesions, the conditions for loudness recruitment do not exist.

More otosclerotic ears in a better state of preservation and in more advanced stages of the disease must be studied before it can be determined whether the underlying pathology for the perception deafness in advanced cases is specific for the disease. The evidence suggests that the degenerative changes are merely an accelerated form of the process occurring in ears without otosclerosis.

It is interesting to speculate as to the nature of the disease process in high tone deafness. A degenerative change involving either the hair cells or spiral ganglion cells independently would not explain how both these elements come to be involved. Even if both hair cells and ganglion cells were involved simultaneously the supporting elements of the organ of Corti should not be affected. Characteristically, however, the organ of Corti is completely missing up to a point where there is an abrupt transition to normal (fig. 9b). It may be, therefore, that the degenerative change directly involves the supporting elements of the organ of Corti and that sensory and neural degeneration are inescapable concomitants of this process. Of course this is pure speculation.

SUMMARY AND CONCLUSIONS

Animal experiments seem to provide sufficient information on the functional and pathologic changes occurring in response to injury to justify the following conclusions:

1. The outer hair cells are more sensitive than inner hair cells to most forms of injury. (An exception may be

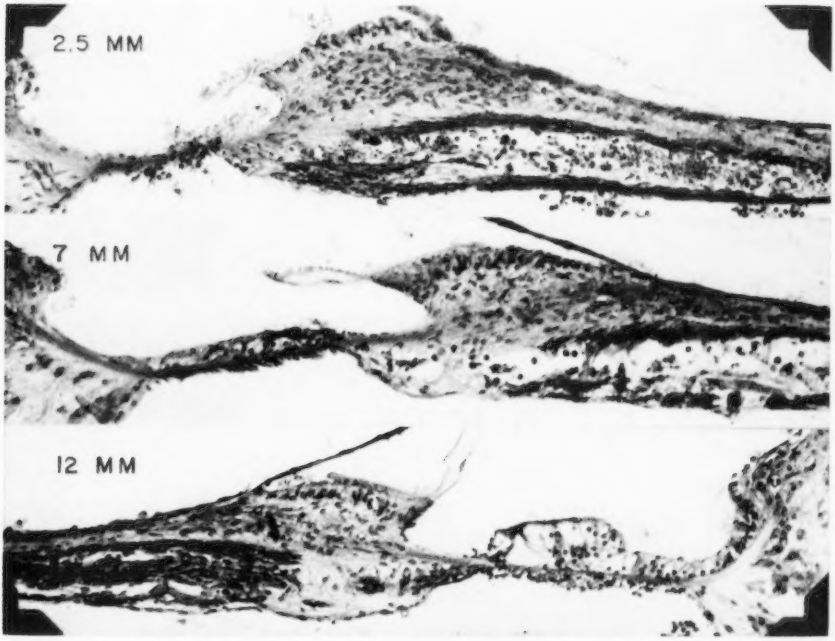


FIG. 9b—Photomicrographs from the 2.5, 7 and 12 mm. areas of the left ear. At the 2.5 mm. and 7 mm. areas the hair cells and peripheral nerve fibers are missing. At 7 mm. the organ of Corti is flattened. At 12 mm. the organ of Corti contains hair cells and normal numbers of nerve fibers.

drug poisoning. See report on streptomycin toxicity by Hawkins and Lurie.[9a])

2. For frequencies up to 5000 c.p.s. a partial loss of outer hair cells creates a deafness of less than 50 db.; a loss of outer hair cells and some inner hair cells creates a deafness of greater than 50 db.

3. Hair cells may degenerate without imposing a degenerative change in the spiral ganglion.

4. In experimental injuries to the organ of Corti it is the severity of pathologic change in the supporting elements, particularly the pillar cells, which best correlates with the degree of degeneration occurring in the spiral ganglion.

5. Primary degeneration of the spiral ganglion produces no change in the organ of Corti or its hair cells.

6. Over 50 per cent of the spiral ganglion cells may be lost in an area of the cochlea without an elevation in threshold for the frequencies having their fields of excitation in that area, providing the hair cells are normal.

Theoretically diplacusis indicates the presence in the cochlea of a localized but severe sensory lesion which has on its basal border a more normal sensory organ. Consequently, frequencies which normally have their point of maximum excitation within unresponsive zones now are able first to excite more sensitive receptors located in a basal direction. The result is a displacement of pitch. This idea is not a new one.[2]

Loudness recruitment seems to require the presence of an incomplete sensory lesion with a normal or near nor-

mal spiral ganglion. In some manner the hair cells which remain seem capable at high intensities of activating not only their own normal nerve supply but also the nerve fibers which are ordinarily excited by activity of the other hair cells.

In human ears with Ménière's disease the spiral ganglion is not different from ears without Ménière's disease. In the early stage of the disease there is little change in hair cell population. Nevertheless, the presence of loudness recruitment in Ménière's disease is strong evidence supporting the idea that the hair cells are affected. The disease probably has its most severe effect on structures in the apex of the cochlea.

The pathologic changes which commonly exist in the basal coil in high tone deafness ordinarily do not provide the necessary requirements for loudness recruitment.

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DISCUSSION

WILLIAM D. NEFF, Ph.D., Chicago, Ill. (by invitation): If I may speak for those of us who are not clinicians, I would say that we feel fortunate in having someone such as Dr. Schuknecht take an active part in research on the basic physiology of hearing. He brings to the experimental laboratory the skills of the otological surgeon plus an intimate knowledge of diseases of the ear. I hope that, as a result of his experimental studies, he may take back to the clinic new ideas which may have practical value in diagnosing and treating diseases of the ear.

The results of the experiments which Dr. Schuknecht has summarized, as well as the findings of other related studies, suggest the possibility of a more complete classification of kinds of deafness. We now commonly speak of conduction deafness and nerve or perception deafness. Dr. Schuknecht has shown the audiograms of experimental animals in which hair cells and nerve fibers were independently destroyed. In some cases, in the clinic, a similar distinction between hearing losses due to receptor cell damage and to nerve damage can probably be made. Perhaps even finer distinctions are possible. If Dr. Schuknecht's hypothesis about the nature of recruitment is correct, one might in some cases differentiate between outer and inner hair cell damage.

I would also like to comment briefly on the problem of assessing accurately the injury or degeneration in the cochleas of animals used in experiments such as those described by Dr. Schuknecht. The findings of numerous studies have shown that temporary and, perhaps, permanent dysfunction may be present in cochleas which appear normal when examined by our present histologic methods. We know, for example, that these methods do not permit us to detect the changes in hair cells or nerve cells which underlie the temporary hearing losses which can be produced by exposure to intense sounds. In interpreting Dr. Schuknecht's results we must keep in mind, then, the possibility that in any given case a somewhat greater region of the inner ear is nonfunctional than that indicated by his plots of nerve cell and hair cell degeneration.

HALLOWELL DAVIS, M.D., University City, Mo. (by invitation): Dr. Meltzer, I am much obliged to you for giving me the opportunity and privilege of discussing this paper because I have tremendous enthusiasm for the material that has been presented, both for the observations and for some of the new ideas that have been expressed. I could pick out a half dozen points that I should like to underline in addition to the very beautiful graphic presentation and the excellent technics that went into obtaining the material in the first place. One is the emphasis that Dr. Schuknecht has given to abnormalities of the supporting structures of the organ of Corti.

I believe that the supporting structures have been "forgotten men" for a long time, and that these cells probably act, in addition to mechanical support, by virtue of certain electrical properties that I believe they have, particularly their electrical resistance. I believe that their resistance to electric current flow may be a determining factor in the sensitivity of the ear. If the resistance is lost, the injured tissue acts as a short circuit for the electrical current flow that should stimulate the nerve.

I want to underline particularly the bit of physiological hypothesis that Dr. Schuknecht offered because it fits precisely with my own thinking, although I cannot prove it any more than I believe Dr. Schuknecht is able to prove it. It is this: *Electrical current flow, for which a small number of remaining functional hair cells is responsible, may stimulate nerve fibers that were connected to other sensory cells that have disappeared.* This is a possible explanation of recruitment.

I hope I have reproduced correctly the ideas that you have in mind, Dr. Schuknecht. I believe that this will be a very important hypothesis in the next several years, to be examined for further support or refutation. I repeat that it falls exactly into my own thinking. The hypothesis that I presented only a month ago before the British Association for the Advancement of Science is, essentially, that generated in the stria vascularis is a powerhouse which generates an electric potential essentially and that the flow of current is modulated by the hair cells. The resulting fluctuations in current flow are the electrical stimulus to the nerve fibers attached to the hair cells. This picture, I think, makes plausible the suggestion that Dr. Schuknecht has laid before you, and his suggestion as far as I know has not been specifically and clearly formulated previously.

FOR THE GOOD OF OTOLOGY

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EVER since Schwartze first described the simple mastoidectomy for acute suppurative mastoiditis and the Schwartze-Stacke radical mastoidectomy for otherwise incurable chronic middle ear suppuration, and up to the advent of the sulfa drugs and the antibiotics in 1937 and 1939, the practice of otology consisted mainly of the surgical treatment of a middle ear abscess and the complications resulting therefrom. During that period there were great contributions made in clinical diagnosis and histologic studies of temporal bone disease which stimulated the progressive evolution of the surgical treatment of temporal bone infection and the various complications which often accompanied it.

In 1927 an article was submitted to the *Archives of Otolaryngology*. The Editor-in-Chief, Dr. George Shambaugh, Sr., in accepting for publication my manuscript entitled "Simple Subcortical Mastoidectomy," stated in his letter to me dated August 25, 1927:

> It represents the type of contribution for which we would like to reserve the Archives, that is an original piece of work.

The publication of this paper[14] laid the foundation for the gradual development of today's new modern temporal bone surgery. The endaural approach to all temporal bone surgery, which I[9] described in 1938, has been almost universally adopted since the advent of the antibiotics.

From Lempert Institute of Otology and Lempert Research Foundation, Inc.
Presented at the meeting of the Otosclerosis Study Group, Oct. 11, 1952, Chicago, Ill.

There were, prior to the antibiotic therapy period, a number of great otologists in the world, and every big city could boast of otologists capable of doing good temporal bone surgery. However, the fact remains that too many otorhinolaryngologists doing temporal bone surgery in those days were very poorly trained, and some were doing this surgery without any training at all. The few greatly experienced and extremely capable otologists in each large community, outside of capably handling their own temporal bone surgery, were also acting as consultants, bystanders and trouble-shooters for the poorly trained and incompetent men who, unfortunately, were doing most of the temporal bone surgery. Those were—as many of yesterday's otorhinolaryngologists poorly trained in otology refer to it today—"the good old days" in otology. Those were "the good old days" when anyone untrained or poorly trained in temporal bone surgery would not hesitate to attempt this surgery since he was always able to blame the offending streptococcus instead of his own incompetence for his unfortunate sequelae and poor results.

Those were the days when after having accidentally injured the lateral sinus, instead of surgically controlling the bleeding and continuing to remove the pathologic process, everything within reach was packed into the mastoid wound to stop the bleeding, and the operation was called to a halt. The patient was sent back to his bed to await thrombosis of the lateral sinus, with the poor little streptococcus taking the blame for all sequelae. Those were also

the days when if the facial nerve was accidentally and blindly injured, the poor little nonvisualized facial nerve was accused of occupying the wrong and anomalous position within the temporal bone and reprimanded for having been in the pathway of the chisel. Such accidents often formed the basis for the publication of an article warning against such anomalies. Such publication often created a precedent for other articles excusing many other unlucky men doing temporal bone surgery for the same kind of accident. Those were men doing temporal bone surgery who never knew that they injured the facial nerve unless the anesthetist called their attention to the facial paralysis. They always thought about the facial nerve, spoke about it, dreamed about it, but seldom saw the facial nerve while operating. When the anesthetist called their attention to the fact that the face was crooked, they surmised that they must have somehow and somewhere injured the facial nerve. Those were also the days when the Ballance-Duell Operation for the repair of facial nerve injury was hailed as a great milestone of progress in otologic surgery.

I personally looked upon the enthusiastic reception given the Ballance-Duell Operation as a sad commentary upon the specialty of otology. This was a contribution mainly towards incompetent otology after having accepted badly performed temporal bone surgery as an irreversible fact. A much better contribution to otologic surgery, and especially toward humanity, would have been better training of temporal bone surgeons so that facial nerve injury would become less frequent, and when it did occur, the surgeon would know when, where and how it happened. With such understanding an accidental injury to the facial nerve is, as a rule, easily overcome immediately on the operating table and permanent facial paralysis is avoided.

Unfortunately, those were the days when otologists were always taught ways and means of blindly avoiding injury to this or that anatomic structure while doing temporal bone surgery. They failed to become trained in the art of deliberately exposing to view all the surgical anatomy and thus decreasing the danger of accidental injury to such anatomy.

Those were also the days when, in doing temporal bone surgery, hearing function was often carelessly and unnecessarily sacrificed in the name of saving life. The conservation of hearing was seldom taken into consideration. Radical mastoidectomies were performed and hearing function sacrified when mastoido-atticotomies, without sacrifice of the ossicular chain and the tympanic membrane, would have sufficed for the removal of the pathologic process. Those were also the days when radical mastoidectomies were scheduled and reported, but very seldom radically performed. After opening the temporal bone and exposing to view the pathologic processes within it, the surgeon too frequently failed to remove the pathology for fear of injuring this or that anatomic vital structure which was lying hidden underneath the diseased part.

In those pre-antibiotic days there existed neither medical nor surgical treatment which could possibly improve the hearing in those deafened as a result of otosclerosis. Nevertheless, otorhinolaryngologists fully cognizant of these facts unhesitatingly performed tonsillectomies and septum resections, used x-ray and radium therapy, gave hypodermic injections of all sorts of chemicals, fed patients all sorts of vitamins, inflated the eustachian tubes, massaged the tympanic membranes with electric percussors, etc. They did all this with-

out ever improving the hearing in any of the otosclerotic patients. In defending such practice some of these men, in all sincerity, argued that the reason they treated these people with no hope of improving their hearing was solely because of their belief that one must not permit these people to lose hope of regaining their hearing. Furthermore, they piously stated that by treating these unfortunates they saved them from being victimized by quacks.

Those are the very same otologists who since the advent of the antibiotics, and in spite of the great advances in temporal bone surgery made possible by this advent, are spreading the gospel today that otology is finished. "It is no longer what it used to be," they say. If the facts about much of yesterday's otology became generally known, everybody would say, "Thank God, otology is no longer what it used to be."

Today, young men in medicine, contemplating postgraduate study and preparation for the practice of otorhinolaryngology, are confused. They are being told that since the advent of the sulfa drugs and the antibiotics, otorhinolaryngology is a specialty much contracted and limited in scope. This, they point out, is especially true of otology. They are being warned that temporal bone surgery, which was so prevalent in the days prior to the advent of the antibiotics, is practically nonexistent today. Strangely enough, many of these young men who are unfortunate enough to have served their internship in an institution whose chief in otorhinolaryngology thinks that way cannot help but believe this to be true.

A careful, unbiased and frank analysis of the facts as they existed prior to the advent of the antibiotics, and comparison with the true facts which are responsible for less otologic surgery being done today in certain institutions, will reveal that the shortage today is not in surgical material but rather in surgeons who are properly trained to take advantage of the abundance of temporal bone surgery waiting to be done. The practice of otology, more than ever before, today offers a much wider field of usefulness to the properly trained otologist. Too many otorhinolaryngologists, who prior to the advent of the antibiotics always found plenty of otologic surgery to be done, are today doing little or no otologic surgery. The reason these men are doing less otologic surgery is not because there is less to be done, but rather because today much of the otologic surgery these men practiced for years is no longer needed, and the type of surgery in demand today requires special training which too few care to undertake.

With every new advance in temporal bone surgery the pendulum not only is retarded from swinging in the direction of further progress but is actually pushed in the opposite direction. Yesterday's ultraliberal temporal bone surgeon suddenly becomes the extreme conservative of today. Prior to the advent of the antibiotics, myringotomies were often unnecessarily performed by general practitioners, pediatricians and otorhinolaryngologists. Today, almost none of them permit drainage of the infection to be established by paracentesis, even in the presence of clinical evidence of existing middle ear suppuration and bulging tympanic membrane. They employ antibiotics exclusively, without establishing drainage. As a result of such treatment, recurrent attacks of otitis media are today much more frequent than they were when they were treated with incision and drainage alone. Functional testing of hearing in such cases has led me to believe that if normal hearing function is to be conserved in such ears, humanity would be best served if, in addition to the use of the antibiotics, early drainage of the middle

ear infection through a carefully placed and performed incision of the tympanic membrane by an otologist would also be instituted. Whereas mastoidectomies were often performed too early instead of timely, today otologists treat suppurative mastoiditis with antibiotics without establishing drainage of the infection or removing the focus of infection. They convert the acute suppurative lesion into a subacute one, and by repeating this antibiotic therapy with each acute exacerbation they gradually convert the pathologic lesion into a chronic suppurative lesion with loss of hearing function as well. Humanity would be served best if in the presence of mastoid cell suppuration, removal of the focus of infection would be instituted in addition to the antibiotic therapy. Chronic middle ear infections wtih cholesteatoma are being treated both locally and systemically with sulfa drugs and antibiotics, year in and year out, until labyrinthine fistulas form.

Today, surgical treatment of temporal bone infection is considered superfluous by the otorhinolaryngologist poorly trained in otology, who does not like to assume responsibility for removal of temporal bone pathology without having the streptococcus as an excuse for the sequelae of his surgical accidents. Even suppurative labyrinthitis, brain abscess and meningitis of otitic origin are being treated with the antibiotics without searching for and removing the focus of infection. Such treatment is, as a rule, dangerous.

Though temporal bone surgery for middle ear infection and its complications have been largely reduced because of the advent of sulfa drugs and the antibiotics, the antibiotics instead of sounding the deathknell of otology became the renaissance of the new modern temporal bone surgery. Labyrinthine surgery has been developed to a degree of perfection hitherto not dreamed of. A new tremendous field in temporal bone surgery has thus been made available to the otologist. As a result of the evolutionary development of the Lempert fenestra nov-ovalis operation, millions of people, deafened as a result of otosclerosis, can now have their hearing restored and permanently maintained thereafter.

This great advance in temporal bone surgery could not possibly have been reached were it not for the advent of sulfa drugs and the antibiotics which permitted experimental surgery in the human without danger of postoperative infection. However, this middle and inner ear surgery is technically so delicate that only those specially trained in this surgery can hope to do it successfully. Unfortunately, there are not enough otologists today who are sufficiently trained to do successfully the constantly tremendous amount of fenestration surgery. Many otologists have abstained from being trained in this new surgery because they felt that in many cases where a hearing improvement was originally obtained following the fenestration operation, it was soon lost again because of osteogenetic closure of the newly created fenestra. This long-existing obstacle to the permanent success of fenestration surgery has finally been removed.

During the last fifteen years all the greatest obstacles to the universal acceptance of the fenestration operation by both the otologist and the hard of hearing people have been conquered. This has been accomplished by developing, successfully employing, describing and teaching for the first time in the history of otology four basic surgicotechnical principles upon which the permanently successful end results following labyrinthine fenestration for clinical otosclerosis depend.

FIG. 1.—Fenestra was made in the external semicircular canal posterior to its ampullated end.

BASIC SURGICOTECHNICAL PRINCIPLES OF FENESTRATION SURGERY

The One-Stage Exteriorized Endaural Fenestration Technic to Replace the Three-Stage Postauricular Exteriorized Sourdille Technic

The publication of this practical one-stage technic[11] rendered fenestration surgery acceptable by both the otologists and the hard of hearing patients (fig.1).

The Bone-Dust-Free Cupola Technic For Creating the Fenestra

This technic[8] permits exposing to view the membranous labyrinth in situ without subjecting it to trauma or injury otherwise sustained by the necessary removal of bone chips which unavoidably enter the perilymph space when the old pulverization technic is practiced. By employing this bone-dust-free technic I have reduced the postoperative inflammatory reaction of the membranous labyrinth to an absolute minimum. The incidence of further impairment of hearing, occurring as an immediate and direct result of the fenestration operation, has thus been reduced to less than 1 per cent. Perilymphatic osteogenesis, formerly resulting from the irretrievable loss of bone splinters within the perilymph space, is thus also avoided (fig. 2).

The Fenestra Nov-Ovalis Technic

During the performance of revisions of osteogenetically closed fenestras following the use of my first described one-stage technic, I have observed and recognized the fact that osteogenetic repair is less likely to occur when the fenestra is made in the surgical dome of

FIG. 2.—The bony cupola is removed intact to uncap the perilymphatic space and expose to view the endolymphatic labyrinth.

FIG. 3.—Fenestra nov-ovalis is made in the surgical dome of the vestibule.

the vestibule, where the fenestra can be made much wider than in the much narrower portion of the external semicircular canal posterior to the ampulla. This resulted in the development and employment of the fenestra nov-ovalis technic.[10] With this technic, after removal of the incus in addition to the head and the neck of the malleus, it became possible to create within the surgical dome of the vestibule a much wider fenestra than by fenestrating into the much narrower and much shallower perilymph space of the external semicircular canal posterior to the ampulla.

The importance of this basic surgicotechnical principle of fenestration surgery soon became obvious as a result of the tremendous increase in the percentage of permanently patent fenestras following its universal adoption by the otologists. However, the explanation for this fact was not yet quite clear to me. Since the only way to prevent ultimate closure of the fenestra gap is to prevent osteogenetic repair of the fenestra rim, I knew no reason why osteogenesis should not take place in the freshly injured rim of a wide fenestra just as readily and just as frequently as in a narrow fenestra. Neither did I understand why, despite the markedly increased percentage of permanently patent fenestras resulting with the exclusive use of the fenestra nov-ovalis technic, about 30 per cent of the fenestras still closed as a result of osteogenesis. This fact convinced me that the creation of a wider fenestra, though obviously important, is in itself not the complete answer to the prevention of osteogenetic closure of the newly created fenestra. The suspicion that some other factor besides the creation of a wider fenestra must be at work in the prevention of osteogenetic closure of the fenestra gap could not therefore be dismissed (fig. 3).

The Invagination Technic for The Deliberate and Consistent Prevention of Osteogenetic Repair and Closure of the Fenestra Nov-Ovalis[7,11-13]

After making the fenestra in the surgical dome of the vestibule and placing the tympanomeatal flap so that it covers and contacts the entire bony region outside the fenestra gap, the portion of the tympanomeatal flap which faces the fenestra gap is invaginated into the gap sufficiently only to contact intramarginally the fenestra rim (figs. 4 and 5).

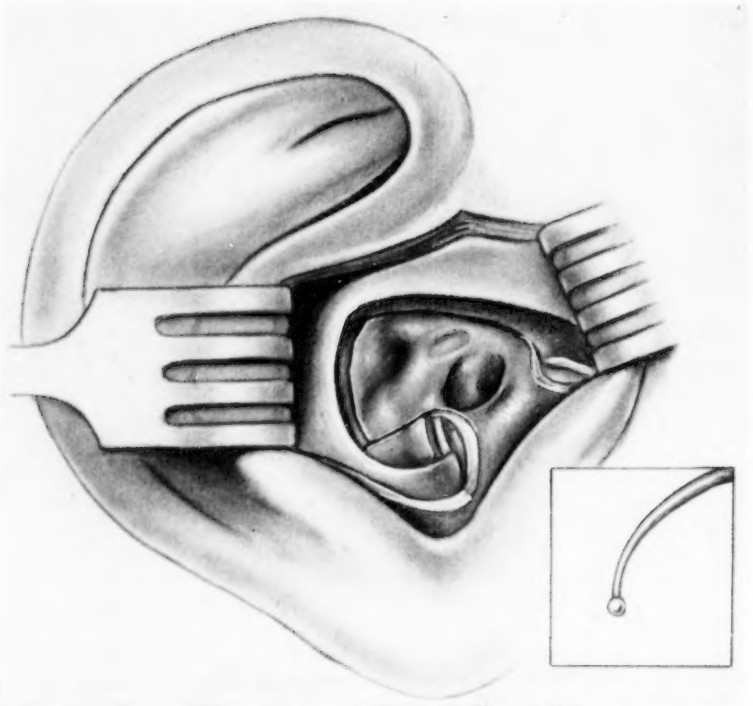

FIG. 4.—Tympanomeatal flap invaginated into fenestra nov-ovalis and the metal invaginator employed for that purpose.

A cotton inlay, moistened in Ringer's solution, is then placed over the invaginated portion of the tympanomeatal flap and molded so that it forces the inner surface to contact, and stay in contact with, the fenestra rim edges intramarginally. This favors its adhesion intramarginally to the bony fenestra rim. The cotton inlay is secured and held in its position with a piece of paraffin mesh gauze molded over it. The rest of the cavity is packed with paraffin mesh gauze to hold the initial packing in its position.

The theory that invagination of the portion of the tympanomeatal flap which covers the region of the fenestra proper into the fenestra gap is essential for the prevention of osteogenetic closure of the fenestra was first advocated in 1938[11] and reiterated in 1947.[12] This theory was finally indisputably proven as being correct and this proof was published in 1950[7] and in 1951.[13]

As a result of observations made by inspecting a large series of postoperative fenestra regions and comparing and differentiating the findings which could be held responsible for osteogenetic repair having taken place in some fenestras and not in others, I have learned the following facts.

1. Osteogenetic closure of the fenestra gap takes place only when the tympanomeatal flap covering it does not become adherent to the fenestra rim.

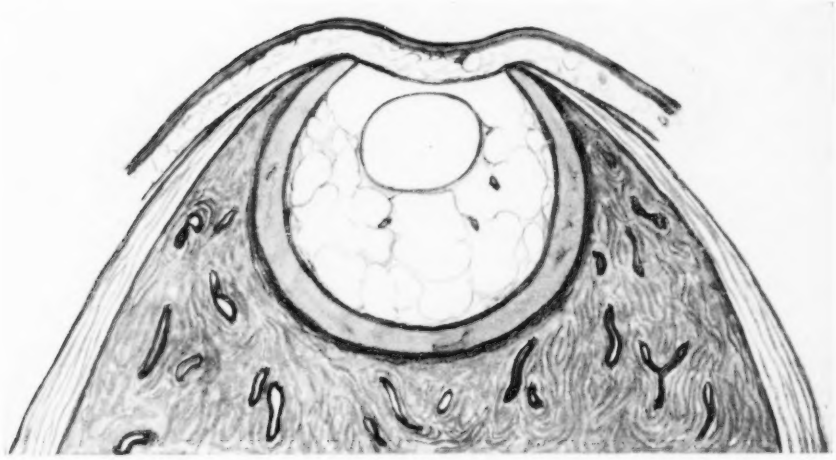

FIG. 5—Diagram showing the cross-section of permanently patent fenestra. Whenever the inner surface of the tympanomeatal flap became invaginated into the fenestra gap and became adherent to the lateral walls of the fenestra rim, osteogenesis was prevented intramarginally within the fenestra rim and the fenestra remained permanently patent.

2. The tympanomeatal flap never becomes adherent to the fenestra rim unless the portion covering the fenestra proper becomes invaginated into the fenestra gap.

3. Whenever the flap became invaginated into the fenestra gap and it became adherent to the fenestra rim, osteogenesis was prevented and the fenestra remained permanently patent.

4. Whenever the flap remained tightly stretched across the fenestra gap, without becoming accidentally invaginated into the gap, osteogenesis was prevented wherever the flap became adherent to the outer surface of the bony capsule but not to the rim of the fenestra gap with which the flap did not come in contact and therefore could not become adherent to it.

5. Accidental unplanned invagination of the tympanomeatal flap into the fenestra gap took place frequently ever since adoption of the fenestra novovalis technic whereby the creation of a much wider fenestra was made possible. This fact suggested that a wider fenestra facilitates invagination of the tympanomeatal flap into the fenestra gap.

6. Accidental unplanned invagination took place only rarely following the use of my first described technic whereby only a very narrow fenestra could possibly be made within the external semicircular canal posterior to its ampulla. This fact suggested that a narrow fenestra hinders invagination of the flap into the fenestra gap.

Following a careful analysis of the above-mentioned facts it became obvious that for the successful prevention of osteogenetic closure of the fenestra gap with the use of my previously suggested invagination theory[11] it is essential that the fenestra be made in the surgical dome of the vestibule, where it can be made much wider than was possible with my first-described and all other previously employed technics for creating a fenestra in the external semicircular canal posterior to its ampulla.

It also became quite obvious that even the widest fenestra made in the

surgical dome of the vestibule will eventually close as a result of osteogenesis if invagination of the tympanomeatal flap into the fenestra gap had not been technically provided for or did not accidentally take place.

It thus became obvious that unless the newly created fenestra is wide enough to permit it, invagination of the flap into the fenestra gap and its adhesion to the fenestra rim cannot be deliberately or accidentally accomplished. By the same token, it also became obvious that unless invagination of the tympanomeatal flap into the fenestra gap and its adhesion to the fenestra rim is either deliberately or accidentally accomplished, osteogenetic closure of the fenestra cannot be prevented. In other words, the deliberate and consistent prevention of osteogenetic closure of the newly created fenestra depends upon the successful accomplishment of two interdependent surgicotechnical factors: (a) the creation of a wide fenestra in the surgical dome of the vestibule and (b) the invagination of the tympanomeatal flap into the wide fenestra gap to permit its adhesion to the bony margins of the fenestra rim.

This surgicotechnical principle for creating a wider fenestra, with the use of the fenestra nov-ovalis technic, has finally proven itself to be the essentially indispensable foundation for the successful execution of my earlier suggested, and ultimately proven, invagination theory for the prevention of osteogenetic repair within the freshly injured fenestra rim and the subsequent closure of the fenestra gap.

These clinical observations in the human postfenestrated ears, which are substantiated photographically, furnished unquestionable and noncontestable proof that my original invagination theory for the prevention of osteogenetic closure of the newly created fenestra was correct.[11]

It thus became obvious that with planned and deliberately executed technic for invaginating the portion of the tympanomeatal flap facing the fenestra gap in the presence of a wide fenestra, osteogenetic repair of the freshly injured fenestra rim could always be prevented and permanent patency of the newly created fenestra should be obtainable in almost one hundred per cent of the fenestrated ears.

These four scientifically arrived at, clinically and histologically proven basic progressive surgicotechnical developments for the improvement of both the immediate and long term end results of the fenestration operation, have been recognized the world over as the greatest of contributions made to fenestration surgery for otosclerosis. Many well-trained otologists are today doing fenestration surgery with my technic and obtaining, as a result, a high percentage of permanent hearing improvements. If more of our otorhinolaryngologists would properly train themselves in modern temporal bone surgery including the fenestration operation, and if men untrained or insufficiently trained in this modern temporal bone surgery would refrain from attempting it and thus ruining the reputation of this surgery in their communities, the demand for temporal bone surgery would jump to much greater heights than in the pre-antibiotic so-called good old days. The real shortage today is not in temporal bone surgery to be done but rather in properly trained temporal bone surgeons capable of doing it.

Many young men contemplating extensive training in modern otologic surgery, who are not yet capable of evaluating what they read in the otologic literature, are today confused by the various suggestions for technical modifications in fenestration surgery. They fail to

realize that otologists doing fenestration surgery successfully with some variations in the endaural incisions, or irrigating continuously or interruptedly for the removal of bone debris, exenterating the mastoid cell structure either partially or completely, using the microscope or any other means of magnification, are nevertheless basically using the Lempert fenestra nov-ovalis technic regardless of the pseudonym under which they are doing this surgery, as Holmgren so wisely stated.[5] Such nonbasic variations of technic are irrelevant to the long term final success of this surgery.

No one can find fault with such harmless nonbasic suggestions for technical variations, confusing as some of them are to the young otolaryngologist. However, when impractical, imaginary and unproven suggestions for technical variations affecting the basic principles upon which the final success of this surgery rests are made, such suggestions cannot be left unchallenged.

When Shambaugh, Jr.,[17] in describing what he calls the Northwestern Technic, publishes colored drawings and shows motion pictures illustrated with animated colored drawings demonstrating how to cover and endosteum-hem the fenestra edges with the perilymphatic endosteum in order to prevent osteogenetic repair of the fenestra, a basic technical principle is involved which may jeopardize the final end result; therefore, such a technical suggestion cannot be left unchallenged. Though he claims he does this only whenever possible, every otologist with a background of experience in fenestration surgery knows (and so must he, able surgeon that he is) that what he calls endosteum-hemming of the fenestra edges is impossible, since endosteum for such purpose is as a rule not obtainable following the pulverization of the endosteal bony layer of the labyrinthine capsule and the removal of the bone slivers from the shredded endosteum and perilymph space. I am also quite certain that he must know, as well as I do, that even if this delicate cobweb-like endosteum were obtainable for this purpose, the retention of it in position would be impossible, since it would disintegrate, dissolve and disappear from the fenestra rim edges as soon as it was covered with the tympanomeatal flap.

Also, Shambaugh's criticism[17] of my bone-dust-free cupola technic for creating the fenestra without pulverizing the bony endosteal layer, by publishing a drawing depicting bone slivers being pushed with the cupola knife into the perilymph space, cannot remain unchallenged. By using the cupola knife like a can opener and first piercing the endosteal bony wall only with the point of the cupola knife and then cutting this thinned-out endosteal bony wall from within outward, instead of from without inward, this does not happen. Even if it should accidentally happen occasionally, one must concede that it is much better than always losing bone slivers in the perilymph space with pulverization.

When Simson Hall and Millar[2] give the impression that the control of bleeding is such a great technical problem in fenestration surgery, by suggesting such heroic and dangerous means as lowering the blood pressure with either arteriotomy or parasympathetic block for obtaining a bloodless field, young otologists who have seen this surgery and are contemplating doing it themselves are confused. The fact is that control of bleeding in fenestration surgery is less of a problem than in any other surgery and never requires more than the ordinary surgical means for soft-tissue control and the polishing burr for its control in bone.

When otologists not trained in the use of the surgical technic for the end-

aural approach to temporal bone surgery advocate using a combination, nonsurgical in principle, of the postauricular and the endaural approach to the fenestration operation, the young otologist is confused.

When Rüedi and his associates,[15] with limited experience in fenestration surgery, express preference for the two-stage technic because they believe that better results can thus be obtained, in spite of the fact that Sourdille's experience with similar technic was most unfavorable, the young otologist is confused.

When otologists hopefully suggest some technical modification of the fenestration operation, eventually find such modification wanting, but fail to report that their suggestion was erroneous and permit themselves to be forever quoted, the young otologist is confused.

When a claim is repeatedly made in the otologic literature that the chorda tympani nerve is capable of hearing function and that by severing it at one end and placing it within the newly created fenestra of an otosclerotic ear, hearing can be restored to the normal decibel level, the young otologist is confused and bewildered.

When Hicguet[3] misrepresents, de Sanson[16] distorts and Simson Hall[1] slants the historical facts about the evolutionary development of fenestration surgery, the young men in otology are confused.

Finally, my friend, the venerable old man, Gunnar Holmgren,[5] one of the great pioneers in the surgery for clinical otosclerosis, stated in 1948, "Lempert's method has conquered," and again in 1950 he stated:[4]

> Julius Lempert's ingenious operative method, at the outset received with considerable skepticism by otologists has in the past five years vindicated itself by rehabilitating, socially and economically, a large number of hard of hearing patients, thereby gaining general recognition.

However, when Holmgren,[6] almost simultaneously with his above-quoted statements, describes and advocates the use of an obviously dangerous but simplified fenestration technic, namely fenestrating the external semicircular canal through a postauricular incision and covering the fenestra with nothing more than neighboring mucoperiosteum, without mentioning his personal experience with such technic, *then I, too, am confused.*

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DISCUSSION

George E. Shambaugh, Jr., M.D., Chicago, Ill.: I wish I had had a chance to read this paper over beforehand. I could have prepared a more intelligent discussion, I am sure. This is really a very pleasant occasion for me, because it is reminiscent of the early days of our Otosclerosis Study Group, when we used to have some very stormy meetings. I am afraid that as we are growing older we are all getting more mellow and the meetings are not as stormy as they used to be.

I subscribe, with very few exceptions, to everything that Dr. Lempert said. There is no question that his contributions to this work are monumental. We all know that. And the operation which we now use is his operation, which some of us have attempted to modify in ways which seemed advantageous to us. Whether our modifications are good or bad, it is a little brash for us to try to say at this time. I think time alone will tell. This applies to the idea of endosteum-hemming, which Dr. Lempert complimented me by mentioning.

I would be the first to claim that it is definitely an advancement. However, it is an idea which we have thought might be worth something and which we are trying, and perhaps as the years go by we will learn whether or not it is worth something or is worthless.

I think there is one thing in Dr. Lempert's presentation to which I should take exception. I hope I don't hurt his feelings because I have a tremendous fondness, admiration and respect for him. I think in a newly developing field like this, it is a mistake to give the impression that one should not have thoughts. I think that all of us doing this work should try to think things through as we go along. We should try to understand our failures and I, for one, have lots of failures. Not as many as I used to have, but a lot more than I wish I had.

I think that while Dr. Lempert is with us he will continue to make tremendous contributions. I am afraid, however, that he won't be with us forever. The time will come when the rest of us will have to start trying to make our own little contributions. While a younger man may be a little confused in reading these varied ideas in the literature, I think it would be a mistake not to have them expressed.

Howard P. House, M.D., Los Angeles, Calif.: Dr. Lempert's contributions to otology are well known and recognized the world over. Through his efforts in the fenestration operation for otosclerosis, an evolution has occurred in the technic of all surgical procedures on the temporal bone which make them practical and workable for the first time.

Medicine and surgery advance through meticulous research carried on by many men of science. Dr. Lempert has spearheaded the stimulus which draws young men to the fascination and self-satisfaction that research provides in the field of otology.

It is true that many articles have been published which have not weathered the sickle of Father Time. However, these articles may well have stimulated the reader to seek more knowledge through research and thereby have often been of value. It is unfortunate that once such an article is published, it is rarely retracted in the literature.

Certainly one should think several times and carefully weigh the value of his work before submitting it for publication. Therein lies the real contribution which Dr. Lempert has provided in this paper.

REDUCTION OF POSTFENESTRATION VERTIGO BY USE OF ISOTHERMAL ISOTONIC IRRIGATION TECHNIC

CLYDE B. LAMP, JR., M.D.
ROCHESTER, MINN.
BY INVITATION

IN THE course of fashioning the fenestra nov-ovalis it has been common practice to wash away the bone dust and blood with saline solution. Postoperative vertigo and frequently a delay in the hearing rise have been taken for granted. When the correctness of using so-called physiologic saline solution to irrigate the open labyrinth was challenged, an interesting study was initiated. The results were found to be significant.

According to the experiments conducted on cats by Aldred, Hallpike and Ledoux,[1] the values for osmotic pressure of the following fluids in terms of their equivalent in grams of sodium chloride per 100 milliliters solution are: endolymph, 1.058; perilymph, 1.046; cerebrospinal fluid, 1.017 and blood, 0.994.

Commercially available physiologic saline solution is 0.9 gm. of sodium chloride per 100 ml. The use of this solution, which is hypotonic as compared to perilymph, sets the stage for endolymphatic hydrops to occur. Could this be the reason for some of the postoperative vertigo? Could this be the reason why the hearing was slow to rise to the maximal postoperative level?

In cases of endolymphatic hydrops, the presence of recruitment frequently has been demonstrated. Recruitment is absent in pure conductive deafness. A series of fenestration cases were examined both before and after operation with the loudness balance test. Recruitment was absent uniformly preoperatively and was almost equally uniformly present postoperatively. The recruitment was observed to be greatest in the early postoperative period and to diminish gradually as the hearing improved. The recruitment was most pronounced in the frequencies which registered the poorest hearing postoperatively. By the time the hearing had reached the maximal postoperative gain, the recruitment had disappeared. These observations supported the hypothesis that endolymphatic hydrops occurs following fenestration. It seems reasonable that the vertigo might largely be due to the same process.

Prior to description of the preparation of a solution to be isotonic with perilymph, a few physicochemical principles will be reviewed.[2,3]

Boyle's law states that "the volume of a fixed mass of a given gas at constant temperature is inversely proportional to the pressure."

Avogadro's hypothesis states that "equal volumes of gases at the same temperature and pressure contain an equal number of molecules."

Charles' law states "the volume of a fixed mass of a gas at constant pressure is increased by 1/273 of its volume at 0° C. for each degree of rise in temperature."

Van't Hoff showed that the diffusion of molecularly dispersed material in liquid systems is approximately in ac-

From the Section of Otolaryngology and Rhinology, Mayo Clinic.

Presented before the meeting of the Otosclerosis Study Group, Oct. 11, 1952, Chicago, Ill.

cordance with the gas laws. His conclusion was that "the osmotic pressure of any substance in solution is the same as it would exert if it were a gas in the same volume as that occupied by the solution." This holds true for dilute solutions where the volume of the solute is small as compared to the volume of the solvent.

Since a gram molecule of gas occupying a volume of 1 liter exerts a pressure of approximately 22.4 atmospheres, a gram molecule weight of solute in a liter of solvent will exert an osmotic pressure of approximately 22.4 atmospheres.

The manner in which the endolymph and perilymph may have different osmotic pressures can be explained by the Donnan membrane equilibria. When a membrane is separating solutions of diffusible solute and solvent, equilibrium will be attained when the concentration of liquid on both sides of the membrane is identical. However, with impermeable ions on one side of the membrane, there will be a greater concentration of particles on that side at equilibrium. Therefore, at equilibrium, a higher osmotic pressure may be present on one side of a membrane than on the other side. To account for the osmotic pressure gradient, one must assume that endolymph contains more nondialyzable particles than does perilymph.

Consider the endolymph to contain 0.994 per cent of mixed electrolytically dissociable salts plus a nondialyzable substance in quantity adequate to explain its greater osmotic pressure. If such is the case, the use of hypertonic saline solution would not effectively combat hydrops because sodium and chloride ions would enter the endolymph to create Donnan equilibrium. The total effect would be to increase the osmotic pressure of endolymph and produce more hydrops. Therefore, a solution suitable for irrigating the opened labyrinth must contain a combination of diffusible and nondiffusible particles in their proper concentrations. If one assumes the concentration of diffusible electrolytes in perilymph to be approximately equal to that of blood, the concentration of the nondiffusible particles should be such as to give the mixture the same osmotic pressure as perilymph. For the nondialyzable substance, sucrose has been chosen because of its molecular size and because it is chemically harmless to cellular structures.

Since dilute solutions obey the gas laws so that $\frac{\text{Pressure} \times \text{Volume}}{\text{Temperature}}$ equals $\frac{\text{pressure} \times \text{volume}}{\text{temperature}}$ obtained with respect to osmotic pressures, temperature control is indicated. In the past, irrigating saline solution was used at room temperature instead of body temperature.

With the foregoing principles in mind, the irrigating solution may be calculated so as to be isotonic with perilymph. First note the following concentration equivalents. Since the molecular weight of sodium chloride is 58.454,

1.046 gm. NaCl/100 ml water equals 0.1789 molar NaCl solution.
0.994 gm. NaCl/100 ml water equals 0.1700 molar NaCl solution.
0.0089 mole/liter difference.

With respect to osmotic pressure, 1 mole of sodium chloride is equivalent to 2 moles of sucrose, because sucrose does not divide into two ions. Therefore, 0.0089 mole of sodium chloride equals 2 x 0.0089 mole of sucrose, or 0.0178

mole. Thus, the solution should contain 0.1700 mole of sodium chloride and 0.0178 mole of sucrose per liter.

Since 342.296 is the molecular weight of sucrose, then 0.0178 molar sucrose solution contains 6.093 gm. of sucrose per liter.

One liter of solution containing 9.94 gm. of sodium chloride and 6.093 gm. of sucrose when used at 37° C. is isotonic with perilymph.

A sterile solution was prepared by this formula. A water bath system was calibrated to warm the solution to such a temperature that after traversing the plastic tubing it reached the ear at exactly 37° C. while the window was being made. In addition, the irrigations in the earlier part of the operation were done with saline solution warmed to body temperature. In the past year, 45 fenestrations performed at the Mayo Clinic were done by this technic.

The results have been very gratifying. There was a definite reduction in the amount of postoperative vertigo and nystagmus. Most patients were up walking within one or two days after operation. Some walked unaided within eight or ten hours of their operation. The Weber test almost uniformly lateralized to the fenestrated ear on the day of operation. By the next day, most patients had only first degree nystagmus toward the unfenestrated ear. Patients having their second fenestration volunteered expression of surprise at how little dizziness there was.

The audiograms done ten to fourteen days after operation revealed an early rise in hearing, especially in the lower and middle frequencies. The loudness balance test showed no recruitment. An occasional case showed postoperative recruitment and delayed hearing rise, but this was definitely less frequent than prior to the employment of this irrigation technic.

There were only 4 failures. In 2 of these cases the results were good initially but later there was closure of the window with bone. The other 2 were cases in which the diagnosis was not incontestibly otosclerosis. In 1 case, deafness may have been due to alcoholism; and in the other, to quinine.

There are individual variations in the osmotic pressure of perilymph. There is probably some difference between the osmotic pressure of cat perilymph and that of the human being. This solution was designed as a starting point, which seemed far closer to being isotonic with perilymph than did 0.9 per cent sodium chloride. Further adjustments in concentration of fluid were planned according to the clinical results. Because the Section of Otolaryngology and Rhinology has been more than satisfied with the results, no changes have been made.

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DISCUSSION

H. L. Williams, M.D., Rochester, Minn.: I think this paper not only demonstrates the benefit of clinical investigation, but it also demonstrates the superior educational opportunities that some of our younger men have had. At least, it is rather difficult for me to go back to these physical and chemical laws and I sometimes doubt if I was ever exposed to these subjects. But fortunately, these younger men still retain a good deal more, I am sure, than I ever had at the peak of my intellectual ability.

I must report that this method of lavage has been extremely satisfactory from the surgical standpoint and that we are going to continue to use this method. From the results that we are getting in fenestration surgery, I believe it is a real advance.

THE PATHOLOGY OF THE NOSE AND PARANASAL SINUSES
II. RHINITIS AND SINUSITIS

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RHINITIS

IN discussing the pathology of the nose and sinuses in relation to allergy, I spent considerable time on the subject of histology, pointing out the connective tissue changes, dwelling mainly on the nonsuppurative lesions (Semenov[12]). Brief reference was made to the cytologic changes that take place in the epithelium, and some mention was made of the subject of infection. In the following pages we shall expand on these subjects, beginning where we left off, with the pathology of rhinitis.

Owing to its ticklish erectile tissue and its lively autonomic nerves, the nasal mucous membrane is capable of a wide variety of moods and disorders.[5,6,15,16] Since allergy, infection and psychosomatic disorders attack it so frequently, and, strange as it may seem, sometimes operate in unison, we must be able to decide almost at a glance whether or not a patient who already is in the throes of one condition is coming down with another. While we have various ways of establishing the differential diagnosis, there is one that is more helpful than the others, namely, the cytologic examination of nasal and sinus secretions, a procedure that is based upon the objective methods of histopathology.

THE CYTOLOGY OF NASAL AND SINUS SECRETIONS

Although the following observations apply mainly to nasal and sinus secretions, the general principles may be applied to other secretions and exudates—pharyngeal, tonsillar, bronchial, aural and salivary gland in origin. Inasmuch as interpretations naturally vary with the histology of the epithelium present in the area under consideration, it may not be out of place to mention the distribution briefly.

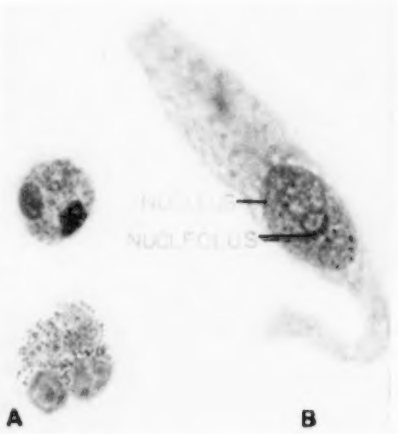

FIG. 1.—Cytology of nasal smear, Hansel's stain.[8] (A) Typical eosinophils showing bilobed nuclei and specific granules which stain red in the cytoplasm. (B) Exfoliated ciliated columnar cell showing typical appearance of nucleus which usually is vesicular and contains fine bluish staining granules. There is a definite nuclear membrane around the nucleus. In well-preserved cells the cilia are clearly visible. This one is beginning to disintegrate.

Pseudostratified ciliated columnar epithelium is found in the nasal sinuses, the nasal cavity, the epipharynx, the eustachian tube, the trachea, the bronchi and a large part of the larynx. A single layer of cuboidal epithelium is found in the middle ear and terminal bronchioles of the lung. Whether the alveoli of the lung are lined by epithelial, endothelial or reticular cells is still a moot question.

Stratified squamous epithelium is found in the nasal vestibule, the ear canal, the oropharynx, the hypopharynx, the esophagus, the larynx, the entire anterior surface and upper half

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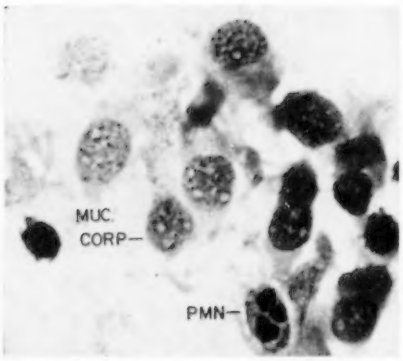

FIG. 2.—Disintegration of the epithelial cells. The cytoplasm disappears and the nucleus remains behind, forming a mucous corpuscle, which often is confused with round cells and lymphocytes. Note the double nucleoli in some of the mucous corpuscles. Compare these cell fragments with the polymorphonuclear neutrophil in the photomicrograph. This smear is from a case of acute rhinitis.

of the posterior surface of the epiglottis, the aryepiglottic folds and the true vocal cords.

Staining of Pus and Secretions

About twenty-five years ago I tried to find a handy, all around stain for smears that would be easy to use, that would bring out as many cytologic details as possible, that would differentiate eosinophils from other granulocytes and pus cells, and at the same time, would stain bacteria. When Hansel perfected his stain, a combination of methylene blue and eosin that looks and acts very much like Wright's blood stain, but is a shade better for our purposes, it lent itself so well to the routine examination of mucus, pus and exudate of all sorts that I now use it routinely and for a great many different purposes, even though it was designed primarily for the identification of eosinophils in nasal and bronchial secretions. Not so long ago, Papanicolaou introduced a stain that is designed primarily for the cytologic details of epithelial cells, particularly the nuclear changes associated with malignancy, but the technic is a complicated one and reserved for special occasions (fig. 2).

Normal secretion. The normal nasal secretion is a clear, watery mucus which, with Hansel's stain, takes a bluish color. A few exfoliated ciliated columnar cells and an occasional lymphocyte and polymorphonuclear leukocyte may be seen here and there, but the mucus is practically acellular under perfectly normal conditions.

Catarrhal inflammation. In catarrhal inflammation we always have an accelerated desquamation of the epithelial cells, and, when the catarrh is a mild one, the nuclei of the cells are well preserved (fig. 3). When the mucus is old or retained for a long period of time, the outlines disintegrate and become hazy.

The leukocytic response in acute inflammation begins with an initial polymorphonuclear infiltration, which is followed almost immediately by an infiltration into the tissues of lymphocytes, eosinophils and macrophages, and within a day or two we may observe nearly all types of cells in the exudate. The picture, however, of a primary infection (normergic reaction) and that of a secondary reinfection (allergic reaction) is not quite the same. In acute infection, or acute rhinitis, a tremendous number of polymorphonuclear cells appear in the nasal discharge, and as the infection becomes more and more severe these cells become more numerous and more immature (fig. 4). The presence of eosinophils in the exudate has come to be associated with a cytologic diagnosis of allergy, but their absence either in tissues or smears is not always conclusive one way or the other. The term "catarrh" (Greek, to run down) merely applies to the discharge that is such a prominent part of the pathology of inflammation of mucous membranes.

In hay fever and nasal allergy, we find, in addition to the mucus and intense desquamation of epithelial cells, a large number of eosinophils in the secretions. When only a few eosinophils are seen scattered about here and there, the diagnosis of allergy is only a tentative one, but when three or four cells are seen in a single field, then one can be fairly certain that an allergic process is present.

Suppurative inflammation. The first step in *pus formation* is a leukocytic infiltration of the tissues (fig. 5). In the

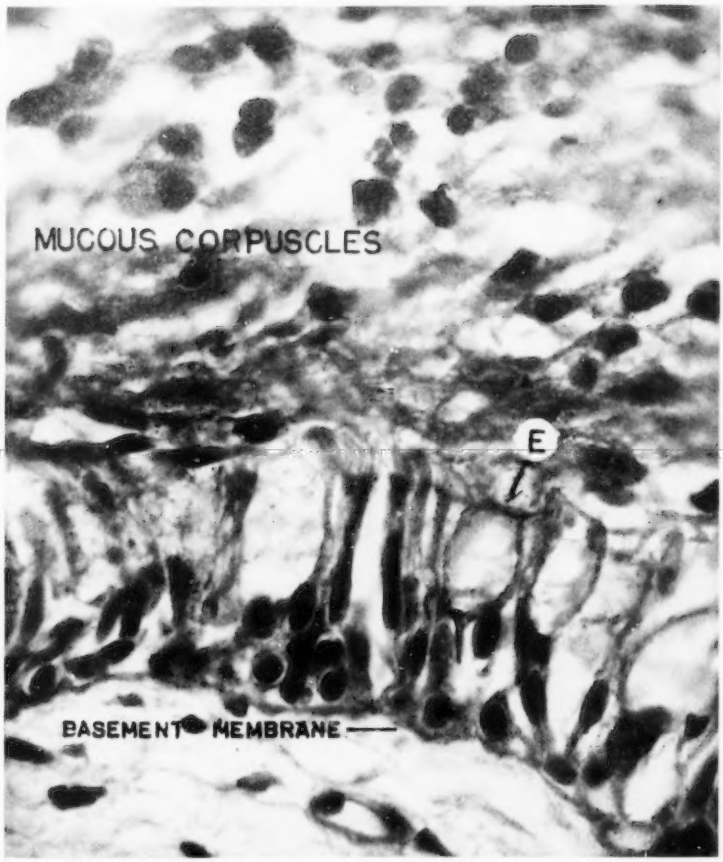

FIG. 3.—Histologic section showing catarrhal epithelium and the shedding of epithelial cells (mucous corpuscles). (E) The line of demarcation between the mucus and epithelium. Note the massive accumulation of desquamated epithelium, the goblet cells, the ciliary insufficiency and epithelial disintegration. In smears, the goblet cells have a ballooned-out vacuolated appearance, and the basal cells have a more rounded shape.

last step, the polymorphonuclear cells disintegrate into pus cells and release enzymes (trypsin) that liquefy not only bacteria but also necrotic tissues. These proteolytic enzymes liquefy desquamated (devitalized) epithelial cells so completely that very few of them can be recognized in frank pus. In catarrhal inflammation, on the other hand, the desquamated epithelial cells always form a prominent and characteristic feature of the disease. As has been stated, in catarrhal inflammation there is no suppuration and necrosis because the reaction is a milder process. In suppurative inflammation there always is some necrosis of the tissues and, of course, pus formation, which means a more severe injury and reaction. With these thoughts in mind, we commonly divide the inflammatory process of the nose, ear and sinuses into three general

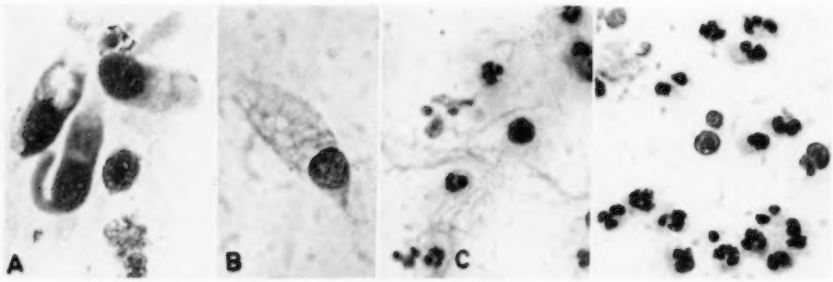

FIG. 4.—(A) Exfoliated columnar cells in nasal smear. (B) Disintegrating cell. (C) Acute rhinitis, typical polymorphonuclear leukocytes and a few lymphocytes.

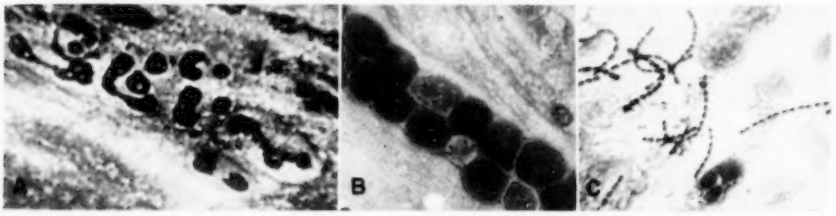

FIG. 5.—(A) Pus cells, band form of nonsegmented neutrophils in the purulent discharge of more severe acute rhinitis. (B) Basal cells and/or lymphocytes. (C) Streptococci in nasal mucus (Hansel's stain).

types, depending upon the appearance of the discharge and condition of the mucous membranes. For example, when we are dealing with the nose and sinuses and consider the type of discharge that is formed during an exudative (purulent) sinusitis, the pathology arbitrarily may be divided into the following types: (1) mucopurulent sinusitis, when the infection is mild or superficial; (2) suppurative sinusitis, when the tissue necrosis is more severe; and (3) gangrenous sinusitis, when the mucosa is practically destroyed.

LEUKOCYTES

It may be well at this point to digress for a moment and briefly review the various leukocytes that appear in the inflammatory lesions of the tissues that we are considering.

Polymorphonuclear neutrophils, the harbingers of pyogenic inflammation, are recognized by their typical multilobulated segmented nuclei, which in the more acute infections assume the immature form of nonsegmented cells. With Hansel's stain the cytoplasm takes a pale neutral color but may, at times, assume a pink color that simulates the appearance of eosinophils. It is extremely important, therefore, to look for the coarse granules that characterize genuine eosinophils.

The polymorphonuclear leukocytes are phagocytes and produce trypsin, a proteolytic enzyme that digests bacteria and devitalized or necrotic tissues, thereby creating abscesses. They are actively motile and emigrate through the walls of the blood vessels only when called for by the tissues, i.e., in response to certain bacteria and toxins.

Under the lower magnifications of the microscope they impart a typical granular look to the exudate, owing to their multilobed nuclei and nuclear fragments.

Eosinophils take a bright red (eosin) stain, owing to their peculiar granules. In making smears the cells frequently rupture and the granules may scatter, appearing as small red dots here and there in the mucus.

These cells typically have bilobed nuclei and distinctive eosinophilic granules. Their function is a mystery, but we do know that they are easily influenced by cortisone. Allergy causes them to migrate in large numbers

into the loose connective tissue and line up beneath the epithelium of the small intestine and respiratory tract. They migrate freely and usually appear in the mucus that forms in the nose or is coughed up in asthma. They are found in the wheals of allergy and may be detected in the fluid shortly after a scratch test is applied to the skin. The granules will stand out in properly stained smears, but the nuclei of these cells often take a pale blue stain. The percentage in a catarrhal discharge, however, is more or less variable because the cells may appear in one slide and not in another, or merely in one part of a single slide. They can be demonstrated in the phlegm that is formed during the height of an attack and sometimes even when the patient is free of symptoms.

A single negative examination, however, does not necessarily exclude the possibility of allergy; accordingly, repeated smears sometimes are necessary before one can establish the diagnosis.

Lymphocytes. In addition to granulocytes, the discharge often contains lymphocytes which may be derived from blood or tissues. These cells take a dark bluish stain and, at times, are called round cells because they and a closely related group of leukocytes all have rounded nuclei and look more or less alike. The nucleus always contains a dark-staining mass of chromatin, which, of course, catches the eye.

These cells are produced in mucous membranes, in tonsils and adenoids, in Peyer's patches and myriads of microscopic foci within the mucous membranes of the nasopharynx, pharynx, larynx, intestines and other parts of the body. Some of the cells remain in situ in the mucous membranes and others emigrate through the tissues and wander farther than any other cell in the body. Being actively motile, they pass easily through the epithelium and the walls of blood vessels. Although lymphocytes vary somewhat in size, some of them being among the smallest cells encountered, the nucleus always is large and prominent and the cytoplasm always is shy and scanty.

Lymphocytic infiltrations of the tissues occur in acute and chronic inflammation. Some of the cells accumulate in small foci or nodules, while others spread out and form a diffuse (scattered) lymphocytic infiltration. Perivascular infiltrations are seen in a variety of chronic conditions, e.g., the collagenous diseases. It is the consensus today that lymphocytes form immune substances and antibodies and that cortisone affects them.

Plasma cells are seen in the tissues rather than in secretions and differ from lymphocytes in the possession of more cytoplasm and a demilune or clear area, in the cytoplasm to one side of the nucleus. They are frequently seen in chronic inflammation, and sometimes develop red acidophilic masses (Russell bodies) in their cytoplasm. Plasma cells have an eccentric nucleus and spokelike chromatin, and along with the lymphocytes they are supposed to form antibodies. The *Russell-Fuchs bodies* are bright red and are supposed to be due to deterioration of the plasma cells. The so-called plasmocytoid body is a plasma cell with closely packed eosinophilic globules.

The lymphocytic plasma-cell reaction, it is said, indicates an optimal antibody production. Excessive doses of antigen seem to stimulate the histiocytes and the antibody response then seems to decline, which, according to some investigators, presumably is evidence that antibody production is a function of the lymphocytes and plasma cells, rather than of the macrophages.

Macrophages look like monocytes; in fact, they are closely related cells, and in tissue cultures monocytes can transform themselves into macrophages. Macrophages are highly motile and highly phagocytic, and in smears they generally are round and have an abundant amount of cytoplasm. They, of course, are typically seen in suppurative inflammation, the cytoplasm loaded with bacteria and debris, the nucleus pushed to one side and indented. They are more common in bronchial secretions than in nasal and sinus secretions.

Macrophages serve many functions. They enter into blood formation and blood destruction, the metabolism of iron, fat, proteins and antibodies and the elimination of a large number of foreign substances. Not all of them behave in the same way or look alike. It is remarkable that the macrophages are mobilized from the connective tissue and not from the circulating blood. In one position the term "resting wandering cell" is used, while in another the term "reticular cell" or "histiocyte" is used. In tuberculous lesions they are called epithelioid cells because they are so large and so cohesive that they simulate epithelium.

The term "macrophage system" includes the entire family (reticuloendothelial system). Metchnikoff, who was the first to describe the entire process of phagocytosis, repeatedly urged that these large mononuclear cells were directly concerned in antibody formation.

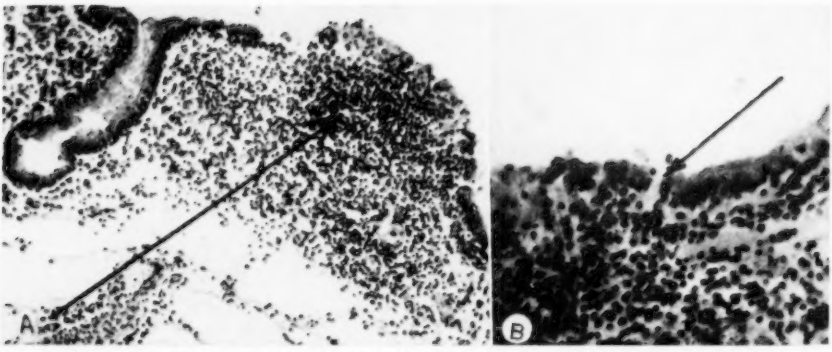

FIG. 6—Histology of chronic purulent sinusitis. (A) Beginning ulceration of the epithelium. Note disorganization of epithelial continuity. (B) Higher magnification showing epithelial and subepithelial leukocytic infiltration, most of which is composed of round cells.

Contrary to the general impression, the predominating leukocytes in the mucous membranes of the nose and sinuses are the lymphocytes, plasma cells and the small round-cell infiltrations. Polymorphonuclear cells seem to come and go so rapidly that very few of them remain in the tissues very long. They migrate through the epithelium, which with ulceration or necrosis begins to lose its continuity (fig. 6) and, as has been stated above, appear in large numbers in stained smears of the mucopus that is formed.

EPITHELIAL DESQUAMATION

As has been stated, catarrhal inflammation, whether allergic or infectious, acute or chronic, always is associated with an abnormal amount of desquamation. In this respect it is not unlike eczema and the dermatologic disorders, in which we see so much shedding and scaling of the epidermis. For example, the epithelial changes in acute rhinitis, or coryza, begin very soon after the onset of symptoms, and while some of the exfoliated cells are living, others are dying and still others are dead and disintegrated into mucoid globules. The changes affect both the columnar cells and the deeper lying basal cells. The shedding of ciliated columnar cells is appreciable within a few hours of the onset of a cold and increases until a peak is reached on the second or third day, then continues until the infection subsides.

Mucous corpuscles, the remains of epithelial cells (basal cells, columnar cells and goblet cells) in which the cytoplasm is lost and only the nucleus remains behind, sometimes look like beaten-up round cells and are easily confused with them. Their nuclei, however, have a different appearance, a vesicular stippled appearance with one or two nucleoli showing (fig. 2). The stippling serves to differentiate these elements from lymphocytes and mononuclear leukocytes, a point worth remembering.

Ciliated columnar cells may occur singly or rolled, by ciliary action, into balls. The cells often are bent in various directions owing to the fact that one end is tapered and the other is broad and wide. The latter sometimes retains its cilia, which in fresh washings continue to beat. The nucleus is oval and stippled with small granules that have a slightly purplish color. The cytoplasm of the cells often contains vacuoles of mucus and hydropic fluid.

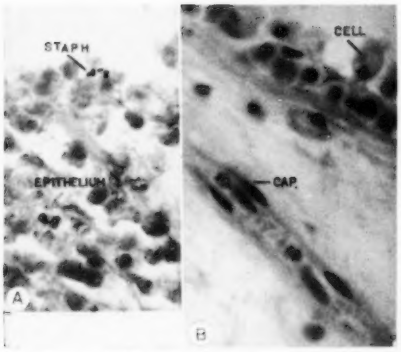

FIG. 7.—(A) Histopathology of suppurative sinusitis, staphylococcus infection and almost complete epithelial disintegration (Case 10). (B) Beginning epithelial regeneration. Note the cuboidal cells (basal cells) and early stratification.

The *basal cells* often are crowded together and have a round nucleus, surrounded by a thin rim of cytoplasm. Their size and shape is fairly constant and they do not vary as much in appearance as do the columnar cells (fig. 5B).

Squamous cells are large and have abundant cytoplasm. The relatively small round nucleus is placed in the center of the cell. These cells may mean metaplasia of the nasal or sinus epithelium,or merely that the suction tube used to collect specimens has been introduced too far into the nasopharynx.

Cancer. It is well to bear in mind that normal epithelial cells eventually loosen and desquamate, that the shedding increases with disease, and that one of the modern methods of early cancer detection is based upon the cytologic examination of smears, using the method of Papanicolaou, which can be applied to any discharge or secretion including otorrhea.[7] The exfoliated cells that arouse suspicion of malignancy, whether they occur singly or in small groups, differ from normal cells in various ways, e.g., size, shape, staining quality and a very irregular appearance. They vary in size (anisocytosis), some of them being much larger than normal (three or four times the size of a normal epithelial cell), for which reason they have been described as giant cells. Some are multinucleated; in others the nuclei are irregular in size or have lost their normal shape and staining qualities, e.g., have become a hyperchromatic (dark staining) dense mass of coarse patchy chromatin. The nucleus often is distorted in shape and may assume a bizarre form, whereas that of a normal cell, even though it may have lobules, is smooth and symmetrical. The size of the nuclei in malignant cells often is out of all proportion to the amount of cytoplasm present and the nucleoli very often are large and prominent, like a "bird's eye." Mitotic figures, however, are not encountered very often in cytologic smears.

Bacteria. Among the various minutiae found in nasal smears, the diverse bacteria, fungi, pollens and particles of dust that may be encountered certainly should be taken into consideration by anyone interested in making a proper diagnosis or, what is just as important, in simply following the course of therapy, much in the same spirit that differential blood counts and smears are made. For example, large numbers of bacteria appear with every cold, but they do not seem to invade the epithelial cells. The viruses, it is said, always invade cells and live and multiply within them. When antibiotics are used, the bacteria disappear from view, which is rather interesting because it helps us make a differential diagnosis between the virus induced nasal inflammations and the bacterial ones (fig. 5C).

POSTNASAL DRIP OR CATARRH

Postnasal drip or catarrh is a common complaint known to the ancients because of the annoying drip of mucus or phlegm into the nasopharynx. One of its causes is allergy, that group of diseases that we now call the collagen diseases, which, interestingly enough, even Hippocrates mentioned in one of his aphorisms (section III, aphorism 31): "To old people dyspnoea, catarrhs accompanied with coughs, dysuria, pains of the joints, nephritis, vertigo, apoplexy, cachexia, pruritis of the whole body, insomnolency, defluxions of the bowels, of the eyes, and of the nose, dimness of sight, cataract, and dullness of hearing." In dealing with this ancient complaint we can learn much, and some-

times but not always effect a cure by understanding the etiology and pathology of the various factors that play a part in bringing it about, and a routine smear and cytologic examination of the discharge always is worth while.

In catarrhal rhinosinusitis there is no suppuration or necrosis. Instead there is a profuse secretion of mucus, which may be watery or of a thick grey or yellowish (mucopurulent) consistency. The color of the nasal mucosa may be the typical bluish white of hay fever and allergy or the dull reddened tint of rhinitis in general, allergic or infectious. The nasal smear in the latter type makes the differential diagnosis for us, and though the leukocytic picture is pathognomonic in some instances, we have much to learn about the cytologic examination of secretions, especially the exfoliated cells, which only within the past ten years have aroused general interest. There are many things that still are unknown about this simple technical procedure. Perhaps in the years to come some of the younger rhinologists will avail themselves of the opportunities for further research in this field, as the Bryan's[1] are now doing.

Half of the people who complain of a chronic postnasal drip have a normal looking nose, and if we take the trouble to examine their nasal secretions we may find eosinophils in about 5 per cent and a normal clear mucus devoid of leukocytes in from 15 to 20 per cent of the cases. On the other hand, when we have positive rhinoscopic signs of inflammation, we may find that 80 per cent of the smears contain neutrophils and that 30 per cent contain eosinophils.

The majority of the cases have some evidence of infection, usually subclinical, and not a little evidence of allergy, which seems to suggest considerable overlapping in a great many instances.

When we take routine sinus washings and study the cytology, after centrifuging the fluid, we will see many mucous corpuscles in the cases of nonsuppurative (hyperplastic) sinusitis, with or without eosinophils in the allergic cases, and definite evidence of pus cells in the infected ones.

The figures cited above apply only to smears and should not be construed as arguments for or against the statistical incidence of either nasal allergy or nasal infection. They are not that good, considering the fact that 25,000,000 or more attacks of acute rhinitis take place in this country each year. Acute rhinitis, coryza and hay fever obviously are seen more frequently than acute sinusitis, and chronic rhinitis also comes to our attention more frequently. This is true first, because of the environmental contacts of the nasal mucosa, a shock tissue of no mean proportions, endowed with an abundance of blood vessels, erectile tissue and glands, particularly over the turbinates, and its exposure to antigens of undisputed potency; second, because of instability of the vasomotor mechanism mentioned above, and, third, because every case of sinusitis has a telltale rhinitis either before or after it.

A latent sinusitis may have nothing more to offer in the way of symptoms and signs than a history of nasal obstruction. The radiographic findings may be minimal, and yet, when such a cavity is irrigated and a relatively small amount of mucopus is removed, the clinical improvement is striking. In some cases, the most painstaking examination is necessary and every available aid, including radiographs, radiopaques, cytologic examination of the discharge and diagnostic puncture and irrigation must be employed before a diagnosis of sinusitis can be established.

Infection of the sinuses usually follows an acute cold or rhinitis which often is forgotten by the patient, who later complains of pain in the teeth, a frontal headache, nasal congestion, nasal discharge, or merely a persistent postnasal catarrh.

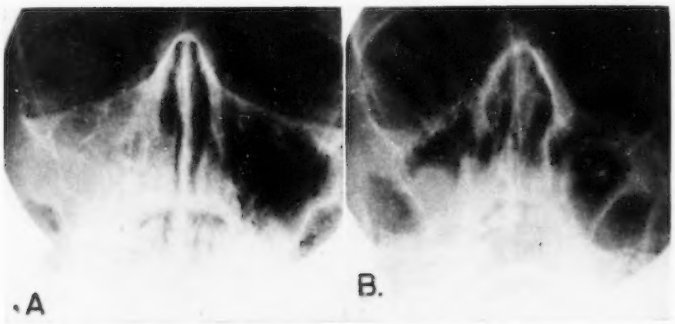

FIG. 8.—(A) Roentgenograms of acute infection, empyema of the antrum, and (B) polypoid swelling (allergic) along the floor of the antrum.

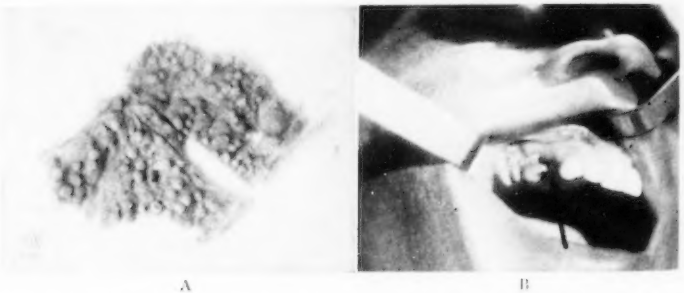

FIG. 9.—(A) Mucous membrane of the frontal sinus showing typical appearance of the granulations in purulent sinusitis. Note the small nodules (granulations) projecting from the surface of the membrane. This specimen was dissected from the cavity and photographed with some enlargement. Compare this gross view with the histology shown in figures 11 and 12. (B) Oro-antral fistula.

SINUSITIS

Though rhinitis and sinusitis have much in common, they are cast in different anatomic molds, and as a consequence, the nasal passages are more susceptible to allergy, autonomic nervous system dysfunction and psychosomatic disorders. The main problem in rhinitis is one of congestion and obstruction; the main problem in sinusitis is one of infection, poor drainage and suppuration. Changed not in kind, but in degree, a nasal infection becomes a different disease when it invades the paranasal sinuses.

The so-called acute exacerbations of chronic sinusitis, as a rule, are combinations of perennial allergy, infection and suppuration. Any one alone is bad enough, but when allergy and infection join forces, a stubborn sinusitis results that is very resistant to treatment.

We must constantly bear in mind that the etiologic factors just mentioned join with certain anatomic factors to create the various pictures seen in everyday practice, in which no two cases are exactly alike. The most obvious type of sinusitis is due to infection alone, or to be more exact, a combination of trauma and infection, such as the purulent sinusitis that occurs when the maxillary antrum is opened inadvertently during the extraction of a tooth, creating an oro-antral

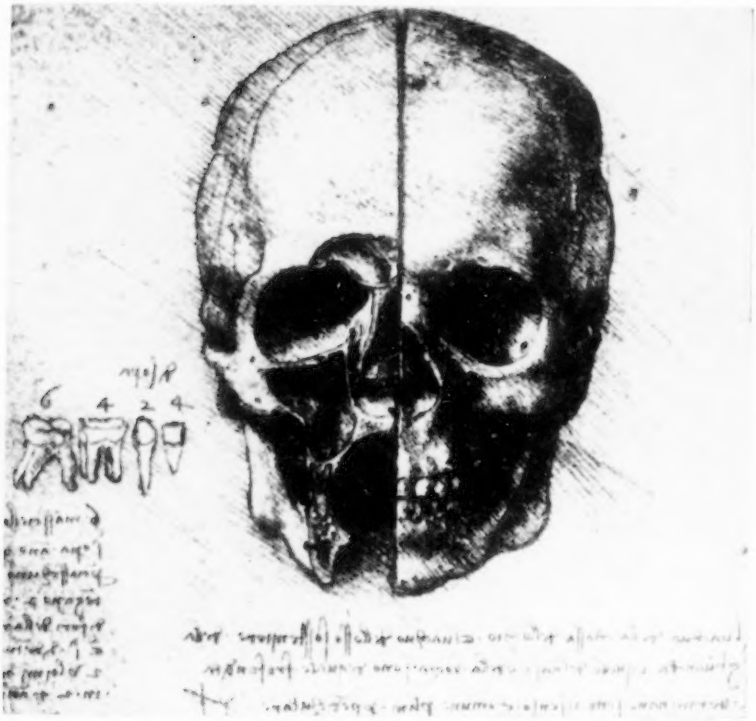

FIG. 10.—Anatomy of the maxillary and frontal sinuses, drawing made by Leonardo da Vinci circa 1489, the first such in history, and still correct. (Courtesy of Royal Library of Windsor Castle and Elmer Belt Library of Vinciana.)

fistula and contamination of the antrum with the bacteria of the mouth.

ETIOLOGY OF SINUSITIS

Precipitating Factors

1. Allergy
2. Virus infections
3. Bacterial invasions, such as acute respiratory infections or from neighboring foci of infection (nasal, pharyngeal, dental)
4. Traumatic with secondary infection, such as penetrating wounds, bullets, chemical irritation and injury

Anatomic Factors

1. Mechanical obstruction and blockade of the nose, such as septal deflections and concha bullosa
2. Poor ventilation and drainage, such as obstructions of the ostia and nasal polyposis
3. Scar tissue and deformity of the sinus outlets, such as that due to trauma or surgery

Contributory Factors

1. Dietary and vitamin deficiencies
2. Lowered resistance associated with blood dyscrasias, anemia, etc.
3. Autonomic and reflex disturbances of the nose, such as vasomotor rhinitis, and psychosomatic disorders
4. Endocrinopathies

Though sinusitis of dental origin was discovered by Nathanial Highmore in the year 1651, the anatomy of the sinuses had been known for more than 150 years by that time, having been first discovered by no less a personage than Leonardo da Vinci, in almost the same year that America was discovered (fig. 10). As a matter of historical interest,

Berengario da Carpi in the year 1521 attributed catarrh to the sphenoid sinuses, and Gabriel Fallopius, in 1562, fully one hundred years before Highmore's great contribution, published a rather amazing amount of information concerning the sinuses.

The *maxillary sinus* by virtue of its size and location is involved in disease more often than the other sinuses. The pathology ranges from suppuration and polyps to cysts and tumors and may be regarded as the prototype of all sinusitis.

When the roots of the upper molar and bicuspid teeth project through the floor into the sinus, a periodontal root abscess may erode the thin plate of bone and infect the antrum. The teeth most apt to be involved are (1) the second bicuspid, (2) the first molar and (3) the second molars.

The bacteria that enter from the mouth produce a characteristic foul-smelling purulent sinusitis, which we may label normergic inflammation in contradistinction to allergic inflammation. In a typical example, the lining membrane is uniformly red, congested and swollen, and in general assumes the soft friable texture of granulation tissue which tends to wall off the infection. The mucoperiosteal lining of the sinuses in this case acts like an abscess wall almost anywhere, and the pathology is that of an abscess in an osseous cavity.

The histopathology of purulent sinusitis has been mentioned previously, and like any acute pyogenic inflammatory process the findings include hyperemia, edema and infiltration of the tissues with pyogenic leukocytes. The surface of the mucous membrane acquires a velvety texture, and in sections we see many small excrescences and crypts in the more chronic cases which are rather typical of chronic infection in mucous membranes (fig. 11). The epithelial injury, whether ulcerative or hyperplastic and metaplastic, seldom is so severe that it is everywhere the same, but rather like a rash or skin disease it tends to be focal, worse in some places than in others. In some areas it is well preserved and in others it is damaged, depending upon the severity of the infection. Leukocytes can be seen emigrating between the epithelial cells, now and then they are fairly numerous, and at some points there may be necrotic masses of epithelial cells, or

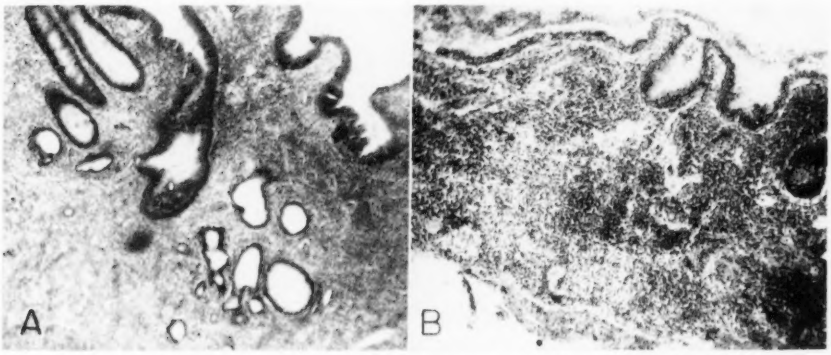

FIG. 11.—(A) Chronic purulent maxillary sinusitis showing cross section of granulations. Note the appearance of the epithelial surface. (B) Frontal sinusitis, the histology is similar in both instances due to the granulation tissue and purulent inflammation and infection.

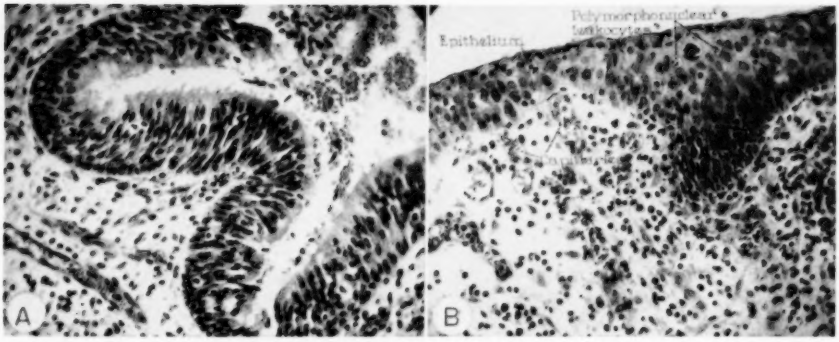

FIG. 12.—Epithelium in purulent sinusitis of one month's duration. (A) In the crypts and troughs between granulations. (B) Over the summit of the granulations. Note the epithelial metaplasia, emigration of polymorphonuclear leukocytes through the epithelium, the round cell infiltration in the subepithelial tissues and the dilated capillaries engorged and congested with blood (x125).

ulcer formation, with a telltale dense accumulation of leukocytes to mark the spot. In the early stages there is no hyperplasia or metaplasia of the epithelium and the infection is confined to the more superficial layers of the mucosa.

As the infection penetrates into the deeper layers of the tunica propria it becomes more serious and the congestion and infiltration of the tissues become more marked, until the lines of demarcation between epithelium, tunica propria and periosteum finally are obliterated by the intense cellular necrosis and reaction. The blood capillaries naturally become greatly engorged and distended with blood, which at times in the more severe hemorrhagic types of infection and inflammation results in petechial hemorrhages. Although there is considerable edema it is not as marked as in allergic processes.

The histopathology of suppuration in the antrum and in the frontal sinus is very similar under the microscope, but quite different clinically.

The anterior ethmoid sinuses usually participate in suppuration of the frontal and maxillary sinuses, creating a combination called anterior sinusitis in contradistinction to posterior sinusitis which involves the posterior ethmoid and sphenoid sinuses. The fact that infection strikes in this manner, picking a single cavity or closely related group, is significant because allergy does just the opposite thing, that is to say, strikes bilaterally and sometimes from head to foot. Another point of interest is the fact that suppurative sinusitis causes such severe symptoms that few persons can endure it for very long; as a consequence the cases that we see today come to us in the early acute

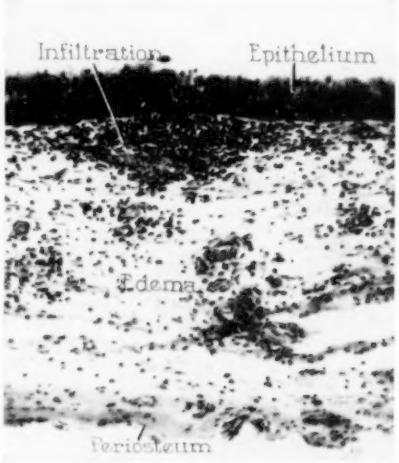

FIG. 13.—Purulent frontal sinusitis of nine months' duration showing subepithelial cellular infiltration, edema of tunica propria and beginning fibrosis of periosteum.

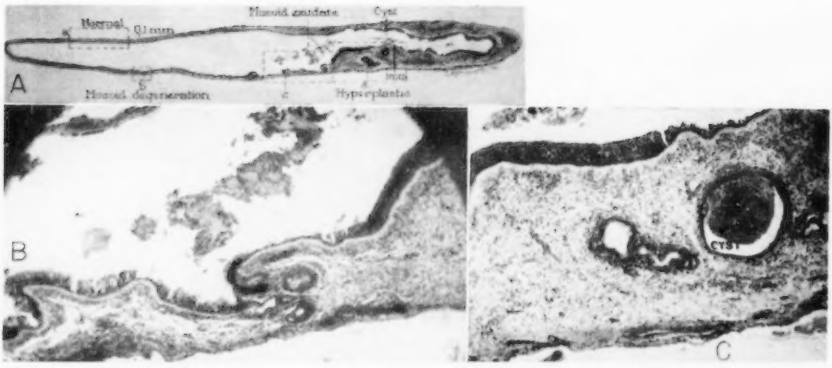

FIG. 14.—Hyperplastic ethmoid sinus membrane (from the same case as figure 13). (A) Cross section of the entire cell. Half of it is normal (0.1 mm. in thickness) and the other half is hyperplastic (1 mm. thick). (B) Transition from catarrhal to (C) hyperplastic pathology. Note the mucoid discharge in (B) and the cyst in (C).

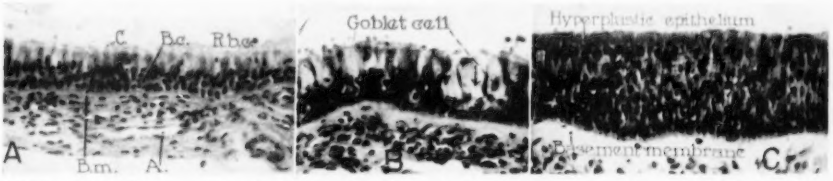

FIG. 15.—Epithelium from the ethmoid cell shown in figure 14. (A) From normal portion of ethmoid. Note the height of the epithelium and thin basement membrane. (B) From the catarrhal area. Note the thickening of the basement membrane. (C) From the hyperplastic area. Note the fact that the epithelium is stratified and by itself is thicker than the entire membrane shown in (A). The pathology here is typical of an allergic sinusitis, eosinophils, etc.

stages of the disease, and only rarely in the chronic stages of suppuration. The situation was different years ago, and still is in some parts of the world even today where modern methods of treatment, antibiotics and adequate drainage of infection are not available to all.

When a sinus infection continues unchecked, areas of ulceration, tissue necrosis, micro-abscesses and gangrene spread and penetrate until the pathology becomes deep seated, invading the bony walls of the sinuses and resulting in osteitis or osteomyelitis. The pathology passes through each stage, going from bad to worse, in an understandable way that we hardly need describe because it is so like all suppurative infections.

As might be expected, there are cases in which an allergic rhinosinusitis is converted to a suppurative sinusitis by infection. The frontal and ethmoid sinuses of such a patient are shown in figures 12, 13 and 15. In this case there is a typical suppurative sinusitis in one cavity and a typical allergic sinusitis in its neighbor, which incidentally shows all of the gradations from a normal area of mucosa to a typically hyperplastic one (fig. 14). The epithelium, interestingly enough, always seems to reflect what is going on beneath it (fig. 15), and as the infectious or allergic process becomes chronic and areas of epithelium here and there begin to undergo hyperplasia, and some-

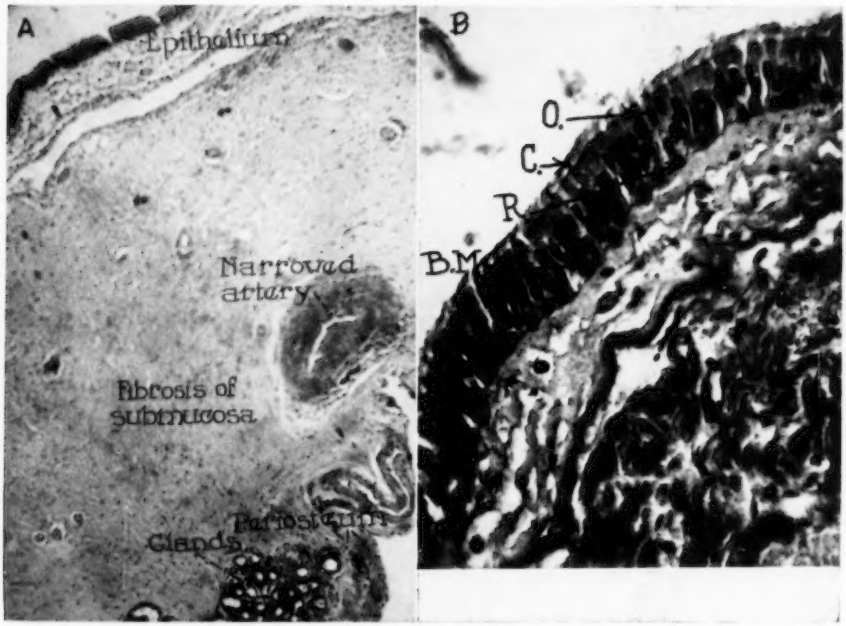

FIG. 16.—Lining of maxillary antrum, thick and fibrous, from a case of chronic sinusitis that got well. (A) Full thickness of the mucoperiosteum showing arteriofibrosis and fibrosis of tunica propria. (B) Perfectly normal ciliated columnar epithelium covers the surface of the membrane everywhere. (O.) Oval nuclei of columnar cells, (C.) cilia, (R.) round nuclei of basal cells, (B.M.) basement membrane.

times metaplasia, the connective tissue begins to proliferate and become fibrotic.

When the inflammation subsides, the tissues may return to a normal state, with only a slight degree of persistent thickening and fibrosis or cicatricial tissue which is not unusual for a purulent process. These membranes, therefore, cast a more dense radiographic picture than the edematous polypoid allergic ones.

We often are asked, "Does chronic sinusitis ever get well?" The answer, of course, is "Yes!" The reason some of us hesitate on this point is because there are no illustrations in the literature showing the microscopic appearances of a sinus mucous membrane after a chronic inflammation has run its course and subsided with complete recovery of the symptoms. An excellent example, which I have reported previously, may as well be reproduced here. Histologic material of this sort is rare because (1) routine postmortem material lacks the necessary clinical data, and (2) routine surgical material is confined of necessity to diseased sinuses.

A man, 50 years of age, had a chronic mucopurulent sinusititis for twenty-five years. All of the symptoms finally subsided, and for a period of four years he was free from colds and nasal discharge, notwithstanding the fact that x-ray examination of the sinuses revealed thickening and blurring of the membranes lining the antrum, ethmoid and sphenoid sinuses. When the mucous membranes were removed and examined macroscopically, they were fibrotic, but when examined under the microscope, the epithelium was perfectly normal (fig. 16). The connective tissue, instead of being loose and areolar, was dense and fibrous, and fully 2 mm. thick. There were no signs of edema or inflammation. The glands of the antral lining were compact and surrounded by firm connective tissue. Similar

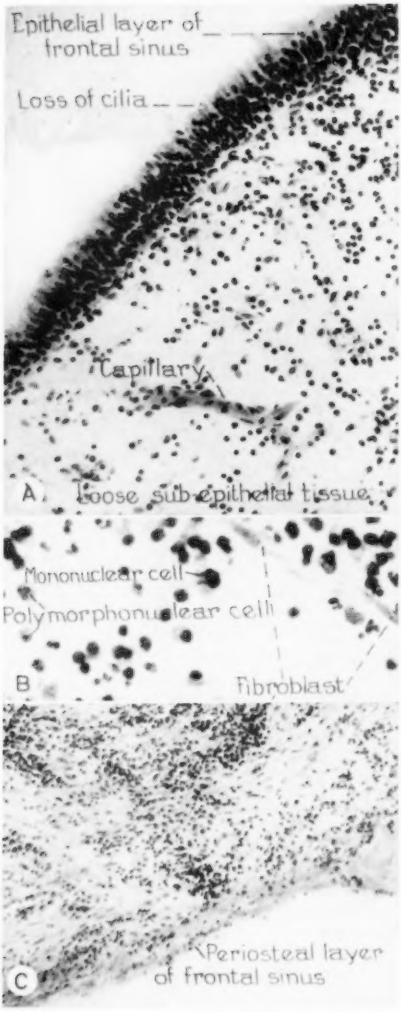

FIG. 17. Chronic recurrent frontal sinusitis, suppurative. This patient had an empyema of the frontal sinus, a severe attack. (A) Loss of cilia, edema and scattered infiltration. (B) Higher magnification showing small round cells and occasional polymorphonuclear cells. (C) The cellular infiltration extends down to periosteum which is fibrotic.

changes were seen in the ethmoid and sphenoid membranes.

As far as the sinuses are concerned, thickening of the lining does not always mean that a disease *is* present, but rather that a disease *was* present. The fibrotic process is a defense reaction, a sign of healing, like scar tissue anywhere (figs. 17 and 18). At times it may include the formation of new bone, but this is uncommon and a rather limited occurrence.

Fibrosis. The fibrosis shown in the cases of sinusitis just mentioned is largely a defense reaction on the part of the collagen tissues and in every instance it is associated with thickening and hyperplasia of the mucoperiosteum.

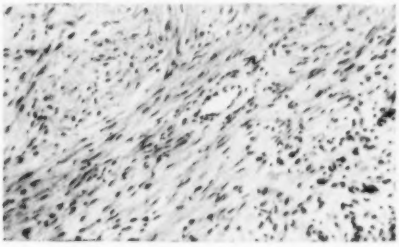

FIG. 18. Fibrosis of the frontal sinus from same case as figure 17.

Those who speak of this condition as "fibrotic sinusitis" may have a point, which, I must say, is valid as long as it is understood that the term is descriptive and not a specific disease of the sinuses, or type of sinusitis. If, on the other hand, this expression is used with the idea that there actually is a specific disease called "atrophic or fibrotic sinusitis," then I must say that I am reminded of the little boy who was asked to discuss the lions on Catalina Island. "There are no lions at Catalina," he said, and sat down. How can we call something that is ten times thicker than normal, which many of these specimens are, atrophic?

Bone. Bone is a living tissue, and more than that, it is endowed with great powers of regeneration and repair, so much so that nearly every broken bone will mend itself and rhinoplastic surgeons can saw away with only one thought in mind—just where and how much to remove. Periosteum shapes bone, supplies it with blood vessels, aids in

the formation of new bone, limiting its development here, causing its resorption there and depositing new bone in another place, all according to natural laws. When the mucoperiosteum is infected, it and the bone connected with it begin to resist, resorting to defense in depth, and thus we may see new bone within a week or ten days after the onset of a suppurative sinusitis. The hyperostosis may be recognized in roentgenograms, the shaggy borders of the sinus cavity or increased sharpness of its outlines being due first, to the fact that the periosteum is more fibrotic and also thickened and, second, to the new bone formation (fig. 19).

FIG. 19—New bone formation, osteomyelitis of the frontal bone. (Atlas of Otolaryngic Pathology, American Academy of Ophthalmology and Otolaryngology, 1942. AFIP 61126. Courtesy Dr. Harris P. Mosher.)

SUPPURATIVE FRONTAL, ETHMOID AND SPHENOID SINUSITIS

The *frontal sinus* becomes important at about the eighth year of life, and in rare instances, at 2 years of age. It is not demonstrable at birth, and sometimes absent even in adults. Suppuration is the main problem of this cavity, with cysts and polyps not nearly as common here as in the antrum.

Before going any further, it may be well to review a few peculiarities of the frontal sinuses. (1) The frontal sinus varies considerably in size; (2) it may be absent altogether; (3) it develops rather late in life; (4) the lining membrane is more rudimentary; (5) the frontal duct is long, tortuous and easily obstructed; (6) radiographs may fail to reveal all of the pathology; and (7) the "union headache"—that come from 10 a.m. to 4 p.m.

Histology. The ciliated columnar epithelium rests on a delicate basement membrane. The subepithelial connective tissue (tunica propria) is thinner than that of the maxillary antrum. The glands are few in number and located near the outlet or frontal duct. The delicate tunica propria blends with the underlying periosteum, forming a mucoperiosteum.

FIG. 20—Lateral view of the sinuses. Note the relationship of the molar teeth and their roots to the maxillary antrum (AFIP 83847, Haymaker[4]).

The periosteal aspect is attached to the bony walls by fibrils of connective tissue, thin-walled capillaries, and blood vessels. The blood vessels pass through the bony walls to anastomose with the circulation of (1) the meninges intracranially, (2) the periosteum externally, and (3) the diploic vessels and veins of the bone (marrow spaces). They offer a natural route for extension of infection—namely, retrograde thrombophlebitis, osteitis and osteomyelitis.

Once an infection reaches a blood vessel, it may (1) enter the vascular channel, or (2) follow the loose, perivascular connective tissue. It works its way into the bone by either method, attacking first the soft connective tissue within the haversian canals, and then the bone itself, and can break through a thin partition more quickly than a thick one, creating in cases of fulminating sinusitis, a microscopic fistula and meningitis almost overnight.

The *sphenoid sinus* develops at approximately 5 years of age and may be pneumatized as early as the first year. Notwithstanding the fact that it is recorded in history as the first of the paranasal sinuses to have been studied seriously and considered as a cause of disease (by Berengario in 1521), sphenoid sinusitis still is the most difficult to diagnose and handle properly.

The *ethmoid sinuses* follow the antrum in the incidence of disease, with hyperplastic polypoid and allergic disorders heading the list. Their histology has been mentioned time and again, and they may be regarded as the keystone of the sinuses. The notion that ethmoid sinusitis is the most common form of sinusitis, however, is no longer held to be true by most modern rhinologists.

Because of proximity to the eye and the brain, the complications of frontal, ethmoid or sphenoid sinusitis include osteomyelitis (fig. 21), orbital cellulitis, cavernous sinus thrombosis, retrobulbar neuritis, meningitis and brain abscess. A neglected suppuration or empyema in any sinus is a serious threat to life, but the upper sinuses are responsible for most of the deaths. In the following pages, a number of case reports selected from a series of cases seen between the years 1928 and 1938, in the days before antibiotics, may prove to be interesting. Most of them were seen at a large county hospital, and the statistics of the intracranial complications that we saw there have already been reported by Courville and Rosenvold.[2] Among others Kramer and Som[9] and Simpson[13] have reported similar cases.

At that time the treatment of brain abscess had a mortality of 50 per cent; today, with antibiotics and newer methods of treatment, it has been reduced in some quarters to approximately 15 per cent. Subdural empyema and brain abscess still is a serious subject and the current literature stresses the importance of early diagnosis and treatment (Pitts,[11] McCaskey[10] and others). The victim may become mentally confused and lapse into a coma, with death supervening within a few hours owing to the effects of pressure upon the midbrain, pons and brain stem functions. The disturbances in pulse rate, heart and respiratory rate, the convulsions, meaning of dilatation of the pupils and other significant symptoms are beyond the limits of my assignment here, but a few reminders may serve a useful purpose.

In the ten-year period mentioned above, I saw 28 cases of sinusitis die of intracranial complications: frontal sinusitis, 10 cases; sphenoid sinusitis, 10 cases; ethmoid sinusitis, 6 cases; and maxillary sinusitis, 2 cases.

Intracranial Complications

Suppuration of the sinuses always requires adequate drainage, either spontaneous or induced, otherwise serious complications ensue. Though the antibiotics work miracles, they are not infallible, and when dealing with an empyema of the sinuses it is far better to know what to expect before the intracranial complications have developed than afterwards.

The lessons learned through the years are so fundamental to the practice of medicine that few who have ever witnessed the fury and horrors of meningitis, brain abscesses or septicemia can ever forget them.

Suppurative otorhinologic infections invade the intracranial cavity by two routes: (1) direct erosion of bone, and (2) retrograde thrombophlebitis.

Roughly 60 per cent of the cases have gross bone destruction, while 40 per cent merely have a microscopic fistula. A small fistula or erosion through the wall of the frontal sinus, or the roof of the ethmoid sinus, or sphenoid sinus, is more serious than a large one because it is not as easily discovered.

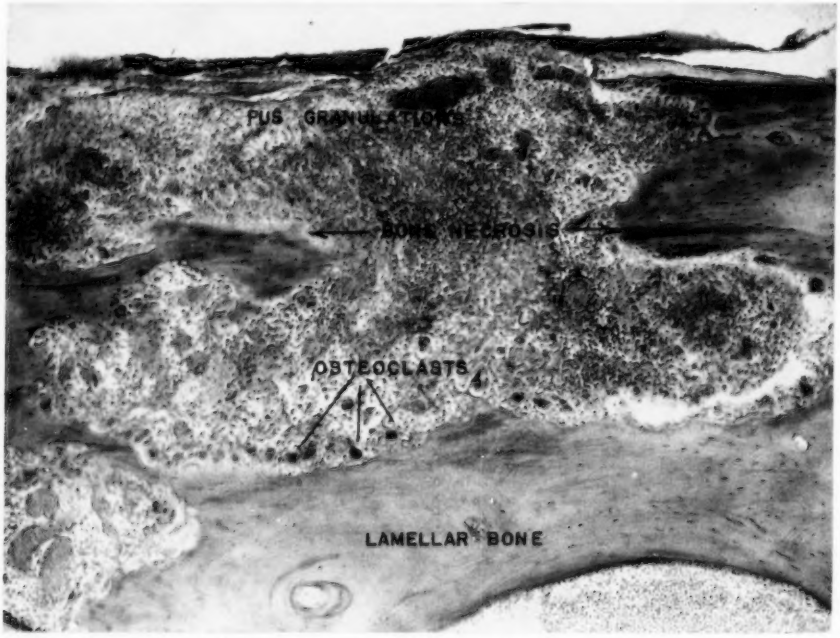

FIG. 21. Osteomyelitis of the frontal bone. (Atlas of Otolaryngic Pathology, American Academy of Ophthalmology and Otolaryngology, 1942. AFIP 61125. Courtesy Dr. Harris P. Mosher.)

The dura mater (internal periosteum) is tough enough to act as a mechanical barrier, accordingly an extradural abscess (collection of pus between the bone and dura) is not as serious as a subdural one. Once the infection breaks through the dura, the prognosis becomes grave.

Meningitis is characterized by hyperemia, reddening and roughing of the meninges, increased formation of cerebrospinal fluid, swelling of the brain, the formation of pus in the subarachnoid space, and an increase in the cell count of the cerebrospinal fluid.

According to Turner and Reynolds,[14] the pathways of infection to the intracranial cavity are as follows:

1. Through the bone (infection due to trauma, skull fracture or instrumentation)
2. Through the blood stream
   a. Arterial blood (infected emboli), and
   b. Venous blood (septic thrombophlebitis)
3. Through lymphatics (a rather remote possibility)
4. Through special regional pathways
   a. The olfactory perineural sheaths
   b. The pial sheath of the optic nerve
   c. The pia arachnoid membrane covering the auditory nerve
   d. The aqueduct of the cochlea, which links the cerebrospinal fluid, the perilymph fluid and the ductus endolymphaticus
   e. The craniopharyngeal canal, which links the nasopharynx (Rathke's pouch) to the pituitary gland. Obliteration of the canal normally takes place early in embryologic life, but it may persist in about one half of one per cent of persons.

When the brain itself is invaded, the infection follows a blood vessel into the substance of the brain. When an abscess results, it may localize and form a capsule, or expand and rupture into a ventricle, causing a terminal meningitis.

MAXILLARY SINUSITIS

Case 1, a man aged 56 years, complained of pain and swelling of the roof of the mouth for six weeks and hay fever for three years.

Examination. Right side of face swollen, marked edema of right eyelid, marked obstruction to breathing, and necrosis of the posterior portion of the hard palate.

RBC	1,400,000
Hb.	30%
C. I.	1.07
WBC	7,100
PMN	29%
Lymph.	26%
Mono.	22%
Stab.	7%
Meta.	7%

The eye was hard and the upper and lower lids indurated (fig. 22). The nose was narrow, the septum thick, and the mucosa exceedingly edematous. The hard palate (floor of the antrum) bulged, and a greenish sequestrum of necrotic bone was found at the junction of the hard and soft palates, creating a fistula in the region of the descending palatine artery which communicated with the antrum.

The right maxillary sinus was opened through the canine fossa. A gangrenous mucous membrane was found. The antrum was filled with desiccated yellow pus. The palatine artery was thrombosed and the osseous channel eroded. The bony walls of the antrum, the canine fossa, the lateral nasal wall were soft and cut like cheese. The bone was unusually pale in color. The thick dehydrated pus in the antrum was under great pressure and had an extremely foul odor and dark green color. The infarction of the sphenopalatine artery, gangrene and bony softening in this case created an oro-antral fistula. At autopsy, we found a subdural abscess in the right middle fossa immediately overlying the gasserian ganglion, which was bathed in pus. The cavernous sinus was filled with green foul-smelling pus which involved the floor of the orbit. There was an abscess in the tip of the temporal lobe of the brain. The sphenoid and ethmoid sinuses were filled with foul-smelling pus.

Comment. This case illustrates not only the complications of sinusitis but also the effects that poor general health, anemia, allergy and neglect have on the course of sinus disease. In other words, we must examine the whole patient and treat all of his diseases, his anemia, his allergy and his infection.

OSTEOMYELITIS

Osteomyelitis creeps through bone by two methods: (1) by extension along diploetic veins, which may cause re-

mote metastatic areas of osteomyelitis, and (2) directly from one marrow space into another.

Infection of compact bone is known as osteitis. Infection of spongy bone (diploetic bone) is called osteomyelitis—a more serious condition because the cavities that contain the marrow are involved. In either event, the bone first becomes reddened, then softened or decalcified, and finally necrotic. Histologically, we find osteoclasts and osteoblasts working alongside each other, which should serve to remind us that bone has remarkable powers of healing and regeneration. Although the pus in the diploetic spaces seeps from one space into another, spreading destruction wherever it goes, the granulation tissue of bone is of a special sort and capable of many maneuvers.

As a rule, osteomyelitis attacks young individuals. It is less frequent in older persons because the marrow undergoes fibrosis with age and this helps prevent the spread of infection. Marrow spaces are found in the bone of the forehead above the eyebrow, and also in the diploetic portions of the alveolar process of the maxilla. Once an infection gains a foothold in bone of this sort it becomes serious.

CAVERNOUS SINUS THROMBOSIS

Case 2, woman aged 22 years, had a wisdom tooth extracted, and within three weeks an osteomyelitis of the jaw, swelling of the face, pus oozing from the socket of the tooth, and cavernous sinus thrombosis and meningitis developed (fig. 23).

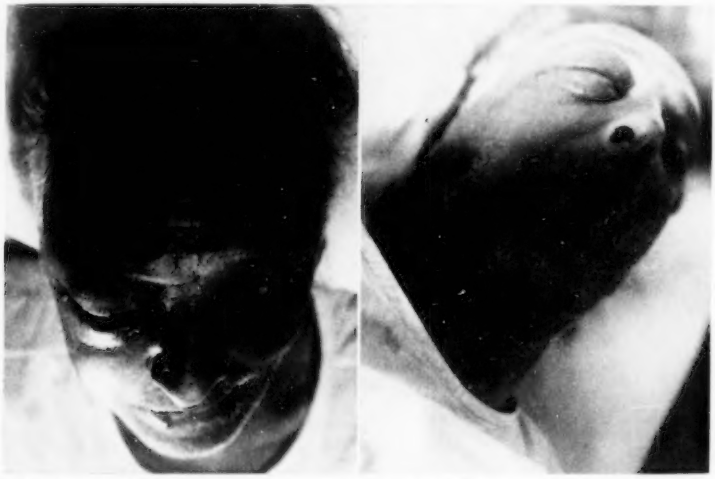

FIG. 22.—Gangrenous maxillary sinusitis with orbital and intracranial complications, case 1. Note swelling of the eyelids and cheek.

At the postmortem examination, we found a fistula in the middle fossa of the skull, near the anterior tip of the left temporal lobe. The osteomyelitis had spread from the maxilla to the great wing of the sphenoid bone, and the cavernous blood sinus was filled with pus. As might be expected, the maxillary, ethmoid and sphenoid sinuses were full of pus and their linings were gangrenous.

SUPPURATIVE FRONTAL SINUSITIS

Case 3, woman aged 37 years, was subject to "colds" and one day complained of a severe frontal headache. Within three days she had a generalized headache, nausea, vomiting and

FIG. 23.—Cavernous sinus thrombosis and intracranial complications due to osteomyelitis of the maxilla, case 2. Note orbital proptosis.

BRAIN ABSCESS

Case 4, child, aged 3½ years, had a suppurative anterior sinusitis, osteomyelitis of the frontal bone and erosion of the anterior ethmoidal roof. The convolutions of the brain were completely flattened over the frontal lobe which was soft and fluctuant, and contained a large abscess (5 cm. in diameter) filled with heavy yellow pus. The abscess had a well-

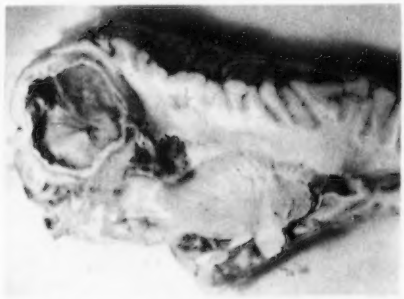

FIG. 24.—Frontal lobe brain abscess, in a child, case 4. The large abscess is encapsulated.

stiffness of the neck. The Kernig test was positive and there was definite evidence of meningitis. The spinal fluid pressure was elevated to 400 mm., the cell count was 4,800 (90 per cent polymorphonuclear leukocytes) and many Gram-positive diplococci were seen in the smears (culture pneumococci).

The floor of the right frontal sinus was tender to touch, the nasal mucous membranes were red and congested but no pus could be seen in the nose. The nasal septum was impacted against the middle turbinate, creating obstruction of the frontal duct and anterior ethmoid area. Radiographically the frontal sinuses were small, and the right one was quite opaque.

An external trephine opening was made in the floor of the frontal sinus. Greatly swollen lining in the nasofrontal duct and empyema were noted. The patient died within a week of the onset, and the postmortem examination disclosed an osteoma about 1 cm. in diameter (attached to the interfrontal septum) which subdivided the cavity and created retention of greenish pus in the posterior portion. She had a subdural abscess on the medial aspect of the right frontal lobe.

organized wall 5 mm. in thickness, and posterior to this encapsulated abscess there was a smaller one with irregular outlines and no wall of encapsulation (fig. 24).

FRONTAL SINUSITIS AND BRAIN ABSCESS

Case 5, boy aged 13 years, had an acute "cold" from which he seemingly recovered. About two weeks later, he began to complain of headache (right forehead, nose and eye), and two days later had swelling of the frontal area and eyelid with exquisite tenderness over the frontal sinus (fig. 25). The nasal mucosa was red and congested, and a greenish postnasal discharge was present on the right side. There was no tenderness of the maxillary sinuses and no pus in the antrum, but the x-ray report read as follows: "The right antrum and ethmoidal cells are opaque. The remaining sinuses appear clear."

The patient was lethargic on the third day of illness and had a suspicious disproportion between temperature and pulse. Fluctuation appeared on the sixth day, indicating the presence of a subperiosteal frontal abscess. When it was opened and the periosteum elevated from the supraorbital ridge, a gush of

FIG. 25.—Acute frontal sinusitis with swelling of the right eyelid and forehead, case 5.

yellow creamy pus (about 5 cc.) escaped from the abscess. The smear contained long chain streptococci.

The floor of the frontal sinus subsequently was trephined. The headache, however, continued, and the temperature remained elevated. The signs of *increased intracranial pressure and meningitis* at the end of a few days included rigidity of the neck on anterior flexion, bilateral Kernig, bilateral Babinski, relative bradycardia (temperature 103, pulse 62) and choked disc; the *spinal fluid* was clear but flowed over the top of the manometer, indicating an increased pressure.

Cells—24 per cu. mm.; 100% polymorphonuclears
Sugar—normal
Globulin—positive
Culture—negative

The diagnosis was right frontal brain abscess, epidural and very likely subdural abscess.

The frontal sinus was reopened and the posterior plate examined for evidence of osteitis. Near the roof and close to the midline we found a suspicious, softened area, and when it was probed a small fistula began oozing pus. The posterior plate was removed and almost an ounce of thick yellow pus drained from the extradural abscess. The dura was red, rough, and covered with granulation in the region of the abscess.

Smears from the abscess contained many long chain streptococci, which on culture proved to be hemolytic streptococci.

The patient regained consciousness immediately afterward, but subsequently lapsed into a coma due to an intracerebral brain abscess. An emergency tap was made in the right frontal lobe and the intracerebral abscess was found. Once more the patient seemed to improve, but again sank into a deep coma and finally died.

At the autopsy, the right frontal sinus was larger than the left one, which was small, empty, and *not infected*. We found multiple brain abscesses, the superior longitudinal sinus filled with a suppurating secondary thrombus, a heavy accumulation of pus over the frontal lobes, and the convolutions flattened (cerebral edema).

The white substance of the right frontal pole, just back of the frontal sinus, contained a smooth-walled cerebral abscess 1 cm. in diameter. This abscess had a distinct wall. The second abscess was found in the cortex of the medial aspect of the brain. It also was sharply outlined but was not as old as the one in front of it. The third abscess had a more recent origin, as indicated by its hemorrhagic appearance and absence of a definite connective tissue wall.

The *left frontal lobe* contained a fourth abscess of large size and quite recent origin, as indicated by the occurrence of hemorrhages in its walls and its ragged, poorly defined border. The substance of the left temporal lobe had a total of seven abscesses, all subcortical.

Summary. (1) Empyema of the right frontal sinus with erosion of the posterior wall. (2) Extradural abscess along the frontal midline. (3) Subdural abscess, at first localized and secondly generalized. (4) Suppurative thrombophlebitis of the superior longitudinal sinus. (5) Infectious thrombosis of the superior cerebral veins, more marked on the left side. (6) Multiple abscesses of the right and left frontal lobes, most of them being due probably to lesions of single vessels, and a large left frontal lobe abscess being secondary to cortical necrosis. (7) Terminal septic meningitis.

Comments. This case is an instructive one and considerable information can be gained from it. In the first place, the infection ran a rapid course and the subdural abscess followed almost immediately after the extradural infection. The osteitis consisted of a minute erosion in the posterior wall of the frontal sinus. The infection simultaneously struck both sides of the falx cerebri and produced an apical subdural abscess very early. This

subsequently broke the primary barriers and spread over the dorsolateral surface of almost the entire cerebral hemispheres, spreading infection along the perivascular spaces via thrombosis of the small cortical arteries and veins, thereby causing a series of multiple abscess cavities. The older abscesses were easily twelve to fourteen days old, while the newer ones were from five to eight days old, indicating very early intracranial involvement, possibly within three or four days of the onset of symptoms of sinusitis. The prognosis was almost hopeless after that.

SINUSITIS AND SWIMMING

Case 6, boy aged 12 years, went swimming with a cold, and ten days later had a frontal sinusitis, with swelling over bridge of nose and forehead which closed the right eye. Temperature, 104 F. Blood analysis: leukocytes, 22,500 with 74 per cent polymorphonuclears. Spinal fluid: cells, 320; polymorphonuclears, 75 per cent, globulin and sugar normal, and culture negative. Right sinus culture indicated Staphylococcus. The patient expired on the seventeenth day.

Summary:

1. Suppurative frontal sinusitis, multiple areas of osteomyelitis.
2. Subdural abscess of the frontal region.
3. Suppurative longitudinal phlebitis.
4. Meningitis and brain abscess. The right frontal sinus and anterior ethmoids contained purulent material. The right antrum also contained purulent exudate but its lining was not thickened.

CHRONIC FRONTAL SINUSITIS

Case 7, man aged 50 years, had a chronic suppurative frontal sinusitis for many years, and symptoms of a subperiosteal abscess for a period of six weeks. He was brought to the hospital in a state of coma and died two days later of meningitis and brain abscess. At autopsy we found (1) chronic osteomyelitis of the left frontal bone, secondary to suppurative frontal sinusitis (streptococci); (2) a large ragged sequestrum in the anterior wall of the frontal sinus, extending from the supraorbital ridge 5 cm. upward into the forehead; (3) the posterior plate completely destroyed; (4) an extradural brain abscess, and (5) an extensive subdural abscess.

OSTEOMYELITIS OF THE SKULL

Case 8, man aged 36 years, had a chronic pansinusitis on the right side. One day a pale swelling appeared over the frontal sinus. It was fluctuant and located above the supraorbital ridge (hair line). The incision along the lower border of the hair line released 4 cc. of pus. Six months later he returned with multiple abscesses in midline (bregmatic region), in left parietal region, in right occipital region, and in right parietotemporal region.

POSTERIOR ETHMOID SINUSITIS

Case 9, child aged 7 years, had a posterior sinusitis and pneumococcic meningitis. One of the posterior ethmoid air cells was filled with a thick, necrotic lining membrane, and the cavity contained thick yellowish green pus. The other paranasal sinuses and middle ears were normal. *Smears* from the sinus and meninges showed enormous numbers of Gram-positive lanceolate diplococci, many with capsules, morphologically pneumococci.

SPHENOID SINUSITIS

Case 10, man aged 45 years, complained of pain over the left side of the head—the ear, the jaw, the side of the face and the parietal-occipital region—for a period of three weeks. The left ear drum was reddened. The nasal membranes were red and congested and purulent discharge was seen in both middle meatuses. He had fever and chills, with a temperature up to 102, and the white blood count was 17,560 (86 per cent polymorphonuclears). The spinal fluid pressure was increased (100 drops per minute), with no globulin or cells. Roentgenograms of the sinuses revealed an increased density of the antra. Although there was no tenderness over the sinuses or mastoid, a myringotomy was performed (only a small amount of thin pus obtained), which is a typical error in cases of sphenoiditis. One week later he had swelling and proptosis of the left eye, and, on the next day, stiffness of the neck. Then a left-sided facial paralysis developed which was followed within a day by coma and death.

Postmortem findings: The diagnosis was fibrinopurulent basilar meningitis, septic cavernous sinus thrombophlebitis, osteomyelitis and necrosis of the sphenoid bone, and empyema of the sphenoid sinuses. When the dura was removed from the middle cranial fossae, a probe was passed, without meeting resistance, into the sphenoid sinus. The roof of the sinus was completely eroded and both cavities were filled with pus. The cavernous blood sinus was filled with purplish pus and clotted blood.

Cultures and smears of the pus produced a pure growth of Staphylococcus albus (see fig. 7A). Microscopic sections of the cavernous sinus revealed a mass of disintegrated

polymorphonuclear leukocytes in a network of fibrin, large mononuclear leukocytes and red blood cells. Pus bathed the left gasserian ganglion and extended over the left petrous temporal bone.

The middle ear and mastoid air cells were clean.

Microscopic sections: The lining membrane of the sphenoidal sinuses was moderately thickened. The deep (periosteal) fibrous connective tissue strands were separated by cellular infiltration. The epithelial lining was completely destroyed and replaced by a thick deposit of fibrin, disintegrated polymorphonuclear leukocytes, mononuclear phagocytes, and exudate. The underlying tissue was heavily infiltrated with plasma cells, small round cells, mononuclear leukocytes and polymorphonuclear leukocytes. The histologic layers were obliterated.

The left ethmoid sinuses were involved in similar pathology.

The right ethmoid sinuses, on the other hand, were lined with a shiny edematous membrane. The epithelium was intact (pseudostratified columnar epithelium). The stroma was edematous and consisted of typical loosely arranged connective tissue with a slight infiltration of small round cells and plasma cells.

SUPPURATIVE AND ALLERGIC SINUSITIS COMBINED

Up until now, we have been dealing mainly with infection. In almost every instance the sinusitis was an acute one, and the patient either recovered or died. Now let us turn to the problem of allergy as it relates to suppurative sinusitis and the complications.

In the first place, it is a chronic process; second, it acts as an obstructive process (polyps); and third, the acute infections that occur lead to the same complications that we have already discussed, but they are more insidious, and often run an up and down course before they finally explode.

ASTHMA, SINUSITIS AND MENINGITIS

Case 11, man aged 59 years, in February 1932, had erysipelas of the face, and an abscess on the left eyelid. Leukocytes, 29,000; polymorphonuclears, 78 per cent; lymphocytes, 20 per cent; eosinophils, 2 per cent. A month later, he had another attack, with swelling and reddening of face, the skin of both cheeks

FIG. 26.—Frontal sinusitis and swelling of the left eye, case 11.

swollen, glossy and dark red, with a definite line of demarcation, and edema of the left eyelid.

In June he had a fever of 101 and pulse of only 76, which casts suspicion of intracranial involvement. His history reads as follows:

Complaint. Swelling of the left eye, 1 day. Frontal headache, 6 days.

Present illness. Has had asthma for ten years, perennial type. The present severe headache over root of nose and left side of the forehead gradually became worse for four days and then slowly subsided. Coincidentally, the left eyelid started to swell (fig. 26).

Previous nasal operations. He has had polyps removed from his nose on many occasions during a period of seven years.

Previous nasal injury. When he was 26 years of age he was kicked in the head by a horse and the scar is still present in his left frontal region, extending down the nose.

Postnasal discharge. Has much phlegm every morning which runs down his throat. It is greyish in color and has been present for ten years.

Examination. Eyeball is proptosed and its movements limited. Tenderness in right frontal sinus. Swelling and redness of glabella. Much swelling and tenderness over the left frontal sinus.

Nose. External deformity of bridge of nose with depression near root—at level of inner canthus of left eye, and evidence of old fracture through left frontal sinus.

Polypi in right side of nose. Resection and absence of the middle turbinate on left, a small atrophic lower turbinate. Pale polypoid membranes. Polypi in the ethmoid region. No maxillary sinus tenderness.

Neck. Not stiff.

X-ray. All sinuses greatly increased in density consistent with pansinusitis.

Blood. 16,400 leukocytes with 80 per cent polymorphonuclear leukocytes.

Diagnosis. (1) Acute suppurative frontal sinusitis and anterior ethmoiditis. (2) Chronic pansinusitis—allergic. (3) Fistula and subperiosteal frontal abscess. (4) Panophthalmitis. (5) Asthma, perennial.

On the eighth day he was in a "quiet delirium," picking at the bedclothes, and had signs of frontal encephalitis.

Spinal fluid. Pressure 320 plus. Greenish yellow color, cloudy. 10,000 cells. Diplococci in smears.

By this time it was too late to do any good, but the only remaining hope was surgery. The bone surrounding the nasofrontal duct was deformed, due to the old fracture. Much pus was present in the upper part of the orbit. It was a purple color, the color of blackberry juce and milk, and came from a fistula in the lateral part of the floor of the frontal sinus.

The lining membrane of the frontal sinus was thick, soft and loosely attached to the bone; at the time of surgery, it was floating in the pus and lifted away without any effort. It was redder than the ordinary polypoid lining of allergy and definitely pyogenic. It was studded with small yellowish abscesses. The anterior and posterior ethmoid sinuses were filled with polypoid membranes, the dull grey, allergic, wet-looking kind. The nasal septum also was polypoid.

Death supervened the next day.

PANSINUSITIS AND MENINGITIS

Case 12, a woman was admitted to the hospital in a coma. She had been complaining of hemicrania on the left side of her head for one week. The pain involved the parietal region, the lateral aspect of the orbit, the temple, and the postauricular and the suboccipital regions, for which she received osteopathic adjustments of the cervical vertebrae three times a day. The day before entering the hospital, she had pain behind the left eye and complained of chilliness, but was able to be up and around, and even went to a motion picture theater. Upon returning home that night, she became irrational and lapsed into coma. About eight hours later she had 2,000 cells in the spinal fluid, but no bacteria were found.

She had had asthma for a number of years, and she blew much heavy mucus from her nose. She never complained of sinus pain, but felt better after her antra were irrigated.

The nose was narrow, especially on the left side. The mucous membrane on this side was red and edematous, and covered with thin mucopus. The septum was deflected to the left, with a large vomerine crest from the tip of the nose to the sphenoid. One could see very little of the middle meatus, and the middle turbinate was impacted and flattened. No polyps were seen. The other side was clear.

The symptoms and signs pointed to sphenoiditis, for which reason the external frontal approach was chosen. The frontal sinus and ethmoid sinuses were filled with polypoid tissue. The sphenoid sinus was full of very soft friable membrane, more necrotic than that of the other sinuses, and about 12 mm. in thickness. Much mucopus was found in all of these sinuses. The bony walls were softened and showed signs of resorption.

Once infection enters the intracranial cavity, the pathology of meningitis is the same in allergic and normergic cases.

Comments. From a clinical point of view, the complications just mentioned have one feature in common. They strike down those who, after years of nasal trouble, lulled into a false sense of security by "the great imitator," are caught off guard.

Normergic inflammation at least strikes head-on, and both doctor and patient behold the full danger; the symptoms and signs are acute and have an abrupt onset. The treatment, which in former years was limited to drainage and general supportive measures, is blessed today with antibiotics. Allergic sinusitis still is a difficult problem, not as grim, perhaps, as it once was, but one that continues to haunt those who must deal with it.

SINUSITIS AND ASTHMATIC BRONCHITIS

The statement has been made, not once but several times, that allergy and infection often combine with one an-

other to create a condition that defies description. Perhaps the best way to describe the situation is to cite another case, an example of the problem that we all have to deal with clinically, but few can follow up as regards to histopathology.

Case 13, a housewife 40 years of age, had a tooth extracted and died immediately afterwards of anaphylactic shock. She had had frequent and severe attacks of asthma for a year preceding her death, complained of a chronic postnasal drip for a period of five years, "hawking up" thick, greenish phlegm, dull frontal headache nearly every day and pain in the region of the right antrum, which she attributed to a tooth.

About three weeks after a severe cold, she had her first attack of asthma. It came on suddenly and was associated with a productive cough, dyspnea and wheezing. The attacks were worse at night, especially during damp weather. Of one thing she was certain, aspirin caused it. (Incidentally, it also has been my experience that when a patient is allergic to aspirin, one can expect reactions of unusual severity.) She had an extensive series of skin tests (200 pollens, foods, etc.), but no positive reactions. The asthma continued unabated for a year.

Rhinologic examination disclosed tenderness over the right frontal sinus, a large polyp in the right middle meatus, the nasal mucosa pink in color, middle turbinate pale and polypoid, and no pus in the nose. Roentgenograms revealed opacity of both antra and a hazy increase of density over both frontals, consistent with sinusitis. A diagnostic puncture and irrigation of the maxillary sinuses was performed; half an ounce of yellow pus was washed from the right antrum and a thick plug of mucus from the left antrum. After this she felt much improved in general, and irrigation of the sinuses was continued at regular intervals for a period of two months. The sinus infection, however, showed no signs of retrogression. On each occasion 5 to 10 cc. of brownish pus was washed out of the right antrum. The diagnosis pointed to a pansinusitis of the allergic type, with purulent right frontal sinusitis and empyema of the right antrum. Because of the empyema and asthmatic bronchitis, sinus surgery was advised.

She was admitted to the hospital and while under observation there, had an attack of asthma that was so severe that she became cyanotic, disoriented, comatose and almost pulseless. Adrenalin, oxygen and artificial respiration plus other heroic measures were used, and at the end of an hour the attack subsided. Two days later she was prepared for sinus surgery with barbiturates, morphine and hyoscine, and I performed a submucous resection of the nasal septum, a polypectomy, right ethmoidectomy and Caldwell-Luc exenteration of the lining of the right antrum under local anesthesia, using cocaine, procaine and adrenalin. The postoperative course was uneventful and she left the hospital on the tenth day. Because of advanced pyorrhea and a chronic abscess of the right upper bicuspid tooth, she went to see the dentist.

The dentist infiltrated the first and second bicuspids with 2 per cent procaine-adrenalin solution and extracted two teeth without difficulty. About five minutes after the extraction, he swabbed the socket with a camphophenol preparation, whereupon she went into a paroxysm of dyspnea, became cyanotic and died within a matter of minutes, notwithstanding the immediate use of adrenalin and oxygen. The attack came on so suddenly and unexpectedly that to this day no one has been able to explain whether it was the phenol or some other compound related to aspirin that precipitated her death. The histopathology of the tissues that were removed from the sinuses at the time of surgery, as well as the postmortem material was studied carefully.

Histopathology. The lining of the maxillary antrum was greatly thickened and polypoid in appearance. It had a yellowish color and measured 3 mm. in thickness. Under the microscope (fig. 27) there was a great deal of edema in the loose connective tissue, much polypoid swelling, hyperplasia of the epithelial layers and underlying stroma, and a chronic small round cell infiltration, with a few eosinophils found here and there in the antral mucosa, which is fairly typical of pyogenic chronic infection superimposed upon allergic sinusitis. The deeper layers of the mucous membrane showed evidence of fibrosis and thickening.

The polyp that was removed from the middle meatus, however, contained a great many more eosinophils. In the presence of allergy, as has been shown previously, tissues removed from one part of the nose may contain a heavy deposit of eosinophils, while tissues removed from another part may lack them, or simply contain a round cell infiltration. The antral lining showed signs mainly of infection. The nasal polyp showed signs mainly of an allergic process.

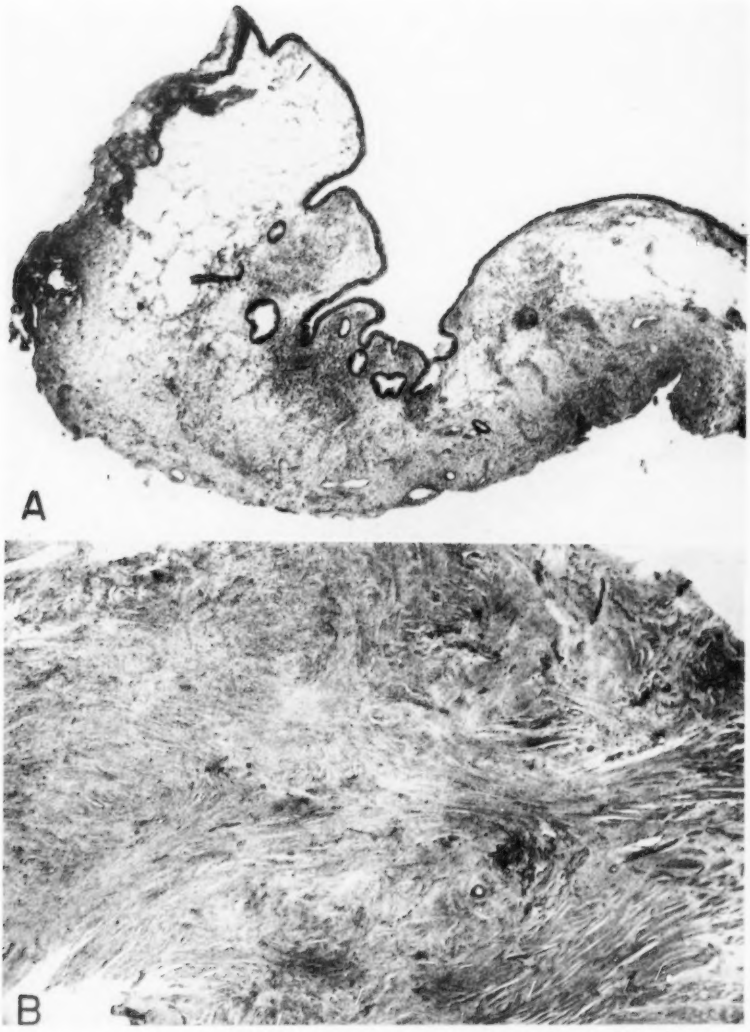

FIG. 27.—(A) Mucous membrane of the antrum removed surgically, showing the typical edema and polypoid swelling of allergic maxillary sinusitis. A purulent, chronic sinusitis was present. (B) Scar tissue removed from the same antrum 2½ months later. It is much thicker than the original membrane. In cases of allergy it is not unusual to find eosinophils and small round cell infiltration in the scar tissue. For this reason, human material is superior to animal specimens. The sinuses of the animals are normal to begin with, while those of the people that we operate on have a disease present. We are interested in the postoperative results obtained in diseased sinuses, not normal sinuses.

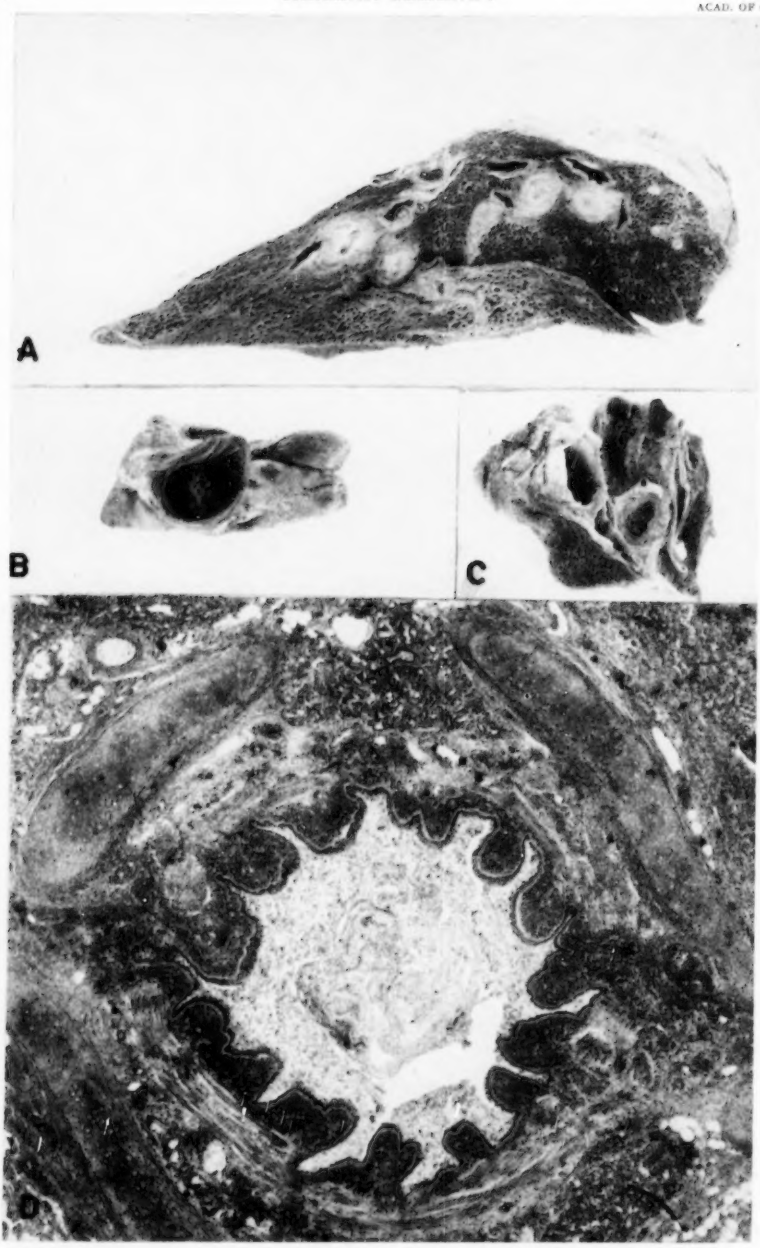

FIG. 28—Pathology of asthma, case 13. (A) Atelectasis of the lung and plugged bronchi. (The oval, whitish areas are plugged bronchi.) (B) Carina, with large plug in the lumen. Viewed from the same angle that one would obtain if he were introducing a bronchoscope. Note the right and left main bronchi and lymph nodes. (C) Pulmonary tissue showing large vein and plugged bronchus. (D) Photomicrograph of a small bronchus. Note the accumulated mucus and folding of the mucous membrane.

During the autopsy, I opened all of the paranasal sinuses, including the sphenoids, which had not been entered at the time of surgery, and found them to be filled with pus. The mucous membranes were greatly thickened in all of the paranasal sinuses, consistent with our previous diagnosis of pansinusitis.

In the two and a half months that had elapsed between sinus surgery and the postmortem examination, the lining of the right antrum, which had been removed at the time of surgery, had become replaced by a thick rough lining composed of scar tissue (fig. 27). No epithelium as yet had entered and covered the surface of the newly formed tissue.

Asthma. The lungs were moderately emphysematous and contained a large area of atelectasis, which undoubtedly was due to bronchial obstruction and was brought about by large plugs of thick mucoid discharge in the large and small bronchi. The plugging of the bronchi was rather striking. The plugged bronchi appear as whitish oval areas in the atelectatic surrounding lung, as shown in figure 28A, a gross photograph. The appearance of the plugged bronchi is shown in figure 28B and C. It will be noted in these illustrations that the walls of the bronchi are greatly thickened; that a thin hyaline ribbon appears just under the epithelium marking the presence of a hyalinized basement membrane, and that the mucosa is thrown into folds, indicating bronchial constriction and spasm. It also is evident from these pictures that the entire mucous membrane lining the bronchial tubes is densely infiltrated with cells, largely eosinophils. Histologically, the bronchi show folded, thickened mucosa, large mucoid plugs in the lumen (Curschmann's spirals) and the usual histologic picture of allergy.

The combined effects of bronchitis, edema, infiltration of the mucous membrane, spasm of the bronchial musculature and plugs of mucoid thick material in the lumen—all of these changes obstruct respiration. The changes that take place in the epithelium include a loss of cilia and this ciliary insufficiency prevents removal of the accumulated debris and secretions much in the same way that it does in the nose and sinuses. For this reason bronchoscopic aspiration sometimes becomes necessary in the treatment of stubborn, intractable asthmatic bronchitis.

Constriction of the lumen of the bronchial tubes. Several factors are concerned in asthma: First, smooth muscle contraction (sphincters); second, thick plugs of mucoid discharge; third, hyperactivity and hypertrophy of the submucous glands; fourth, edema and infiltration of the mucous membranes with or without hyperplasia of the epithelium and underlying stroma; finally, congestion of the blood vessels and capillaries. The smooth muscle fibers, when they contract, diminish the size and lumen of the tubes. The exudate that fills the lumen of the tube may be regarded as a cork. As long as the bronchi are dilated, air can pass around this plug, but when they contract, the tube corks itself.

When a bronchial muscle contracts, it constricts the lumen, thereby reducing the air space. In chronic asthma the bronchial muscle bundles thicken and, with repeated attacks of asthma, hypertrophy. With so much thickening of muscle bundles, epithelial layers, basement membrane, subepithelial connective tissue, desquamation of the epithelium and formation of massive quantities of thick mucus, it is little wonder that the treatment of asthma poses so many problems. Though the fibrosis and hyperplasia of the mucous membranes is irreversible, the vagotonia, spasm, muscular contraction, edema and infiltration of cells, and the hypersecretion of mucus is reversible. These changes are more or less under the influence of sympathetic and parasympathetic nerves that supply the lungs and bronchi. As stated previously, the surface epithelium and formation of mucus at this level is beyond the control of the nervous impulses. The secretion of mucus by the glands that lie in the submucosa, however, is under the influence of the sympathetic nervous system. We are led to the obvious conclusion, therefore, that asthma is due to spasm

of the smaller bronchioles, edema and exudation, and to treat the more severe types we must combine cortisone with some of the older remedies.

As with sinusitis, we may recognize two main types of asthma. The first type is purely allergic in origin and consists mainly of bronchial spasm due to foods, inhalants or pollens. The second type of asthma may be labeled asthmatic bronchitis; in this type there is, in addition to the allergy, definite evidence of infection and inflammation. This type may be due to bacterial infection combined with food and inhalant allergy. The inflammatory lesions may or may not be irreversible in this type, depending upon their degree. Plugging of the bronchi and bronchioles is more apt to occur in the second type. Tenacious mucus and plugging of the bronchi, hyperplasia and thickening of the mucous membrane, hyperplasia of the muscularis and obstruction to respiration lead to emphysema. The bacterial type of asthmatic bronchitis flares up with every acute infection and remains as long as a focus of infection or bacterial allergy continues to act.

The bronchial walls show fibrosis, thickening, exudation and cellular infiltration. The mucous glands become hypertrophied and give rise to a hypersecretion of mucus. The epithelium becomes hyperplastic and thickened and contains a great many goblet cells and mucus-forming cells with a definite loss of cilia. The lesions are very similar to those described in such great detail when we discussed the nose and sinuses. The extensive desquamation of the epithelium, mucus plugs, the hyalinization and thickening of the basement membrane have been described as they occur in the nose and sinuses. The presence of eosinophils in the spirals that form in the bronchial tubes and are coughed up and expectorated, serves to differentiate asthmatic bronchitis from ordinary chronic bronchitis.

Histologically, there is one big difference between the bronchial tubes and the mucous membranes of the nose and sinuses; namely, the presence of muscle tissue in the former. Hypertrophy, hyperplasia and contraction of the muscularis is the one feature that is lacking in the nasal and sinus mucous membranes. Another important difference is the fact that the mucous membrane of the sinuses has large tissue spaces that favor the formation of edematous polyps. The tissue spaces in one case and the musculature in the other constitute the main histologic points to be remembered in discussing the pathology of allergy in the sinuses versus allergy in the bronchi. As regards the nasal turbinates, we must consider erectile tissue, which, as has been stated, is a problem of another kind.

POSTOPERATIVE SCAR TISSUE

According to a recently published (1952) treatise on diseases of the ear, nose and throat, "It has been definitely established that normal, functioning ciliated epithelium can be regenerated following operative removal of the complete lining of the maxillary sinus." The author of the book, or rather the two volumes comprising the set of books, said nothing whatsoever about the scar tissue that forms and is the main problem in nasal and sinus surgery.

Contrary to the impression mentioned above, radical sinus surgery and complete removal of the lining membrane leaves much to be desired in the way of regeneration of a normal functioning mucous membrane. The regeneration myth goes back twenty-five years, to some experimental work on dogs, three dogs to be exact. At that time, I was working with Kistner and had enough human material, at least 16 different

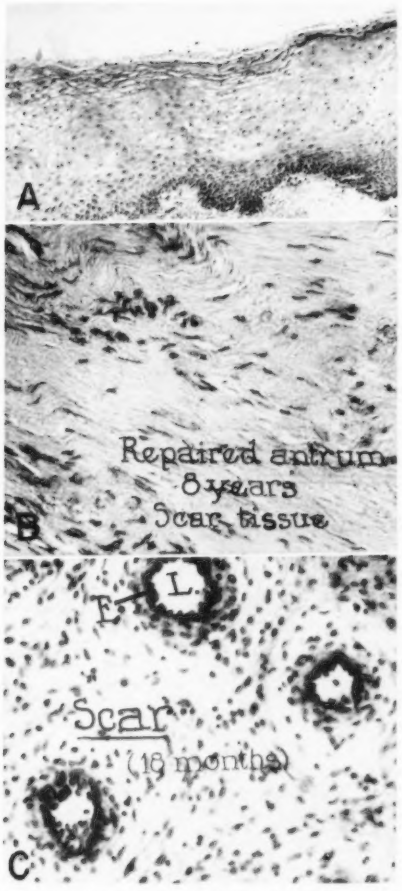

FIG. 29.—Postoperative healing and repair of the antrum. (A) By ingrowth of stratified squamous epithelium. (B) Scar tissue at end of 8 years. (C) At end of 18 months, showing young blood vessels. (E) Endothelium. (L) Lumen of young blood vessels.

specimens from as many different patients who had radical sinus surgery, to be able to draw some definite conclusions which were published and subsequently verified by other competent investigators. This time let us put those three dogs in their graves and bury the ghost once and for all.

When we remove the mucoperiosteal lining membrane that covers the bony walls of the nasal sinuses, it does not regenerate, no more so than the nasal mucous membrane or that of any other organ would regenerate if we removed nearly all of it. The denuded area is repaired, it is true, but the repair is with scar tissue. Depending upon the type of surgery and its extent, the scar becomes epithelialized, casts a dense radiographic shadow, and, if we take samples of this tissue and look at them under the microscope, we may see the various conditions shown in figure 29.

When we compare the thickness with that of a normal sinus lining, we see the disparities that preclude use of the term "regeneration." When we turn a flap of nasal mucosa into the antrum to aid in the epithelialization of the scar tissue, we may, to our great chagrin, learn that the transition to scar tissue is marked by a lack of normal pseudostratified ciliated columnar epithelium (fig. 30).

This does not mean that small areas cannot be quickly repaired by the epithelial cells bordering the area of injury, but rather that in such cases the adjoining basal cells migrate over the denuded area, multiply and dedifferentiate there into pseudostratified ciliated columnar epithelium. The regrowth of epithelium, however, is hampered by the terrain that it must cover, and at times the going is rough. Thus, the underlying granulation tissue and bone spicules raise obstacles and the ingrowth of the epithelium very often is delayed and suffers. The longer it is repressed, the thicker the scar tissue, and the thicker the scar, the more apt it is to act as an hindrance to good drainage, especially in the region of the nasofrontal duct. Curettement is wrong in principle. The scar tissue that results often interferes with drainage, acting as a dam or hindrance across the line of ciliary action. When we dissect carefully and plant a

small mucous membrane flap over the bony wound, the window or duct is more apt to remain open permanently, a highly desirable result.

We all know from experience that scar tissue can produce closure of a sinus no matter how rigid the walls may be, and that the removal of mucous membrane in the region of the ostium results in scar tissue and blockage of drainage. The ostium of the sinus cavity should not be tampered with; when an opening is necessary, it should be made at another point.

Cysts are not uncommon even in scar tissue when the mucous membrane is not removed completely from the bone, especially implantation cysts (fig. 31). In short, disease may result from poor sinus surgery itself, and every surgeon must be absolutely certain that the pathologic condition that follows his technic is no greater than that which he is trying to correct. For this reason, let us not take the word "regeneration" so lightly. Otolaryngology has suffered too much abuse in the past to endure such laxity for long.

The postoperative lining that forms in the sinuses may be thick or thin like any other scar tissue. It is adherent to the bony walls and can only be torn away with difficulty, bringing with it spicules of new-formed bone imbedded in the scar tissue.

The postoperative cavity becomes much smaller in size and often is lined by a layer of scar tissue that is devoid of glands and poorly epithelialized.

The findings vary according to the type of disease present in the sinuses and also the type of operation. For instance, in cases readmitted to surgery we found signs of allergy even in the scar tissue fragments, as well as portions of the original mucous membrane attached to the scar tissue even after radical exenteration.

The nasal mucosa supplies the new epithelium that spreads into the sinus cavity. But where there is a possibility of epithelium extending in from some other source, such as the buccal cavity, via an oro-antral fistula, the new epithelium will be of the stratified squamous variety. When fragments of sinus epithelium remain in the cavity, the regrowth begins with these cells (fig. 29). In any event, the new epithelium may fail to cover all parts of the scar tissue and then crusts ensue.

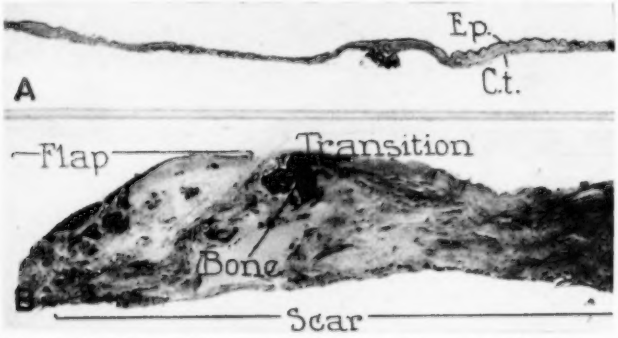

FIG. 30.—(A) Normal thickness of antral mucoperiosteum. (B) Postoperative lining of antrum showing the flap of nasal mucous membrane that was turned into the antrum from the nose. Its characteristic structure ends abruptly and can be differentiated from the scar tissue. After surgical removal of the sinus lining, the bone accepts this scar tissue as its periosteum (x8).

Examination of the tissues filling the surgical opening in the canine fossa, where we could be sure the entire mucous membranes had been removed down to and including the bone, always shows a dense scar covered with a variable cuboidal or columnar epithelium and a complete absence of glands.

When one square centimeter was excised from the nasoantral wall, we found, four months later, a layer of ciliglands; (4) an orderly arrangement of branching arteries and arterioles; and (5) a definite periosteum. But, in the operated portions, we found a very thick mass of fibrous scar tissue, covered by cuboidal epithelium, stratified in some areas and entirely absent in others, the vascularization typical of scar tissue, and no visible glands.

After a complete subperiosteal exenteration of the lining of the antrum,

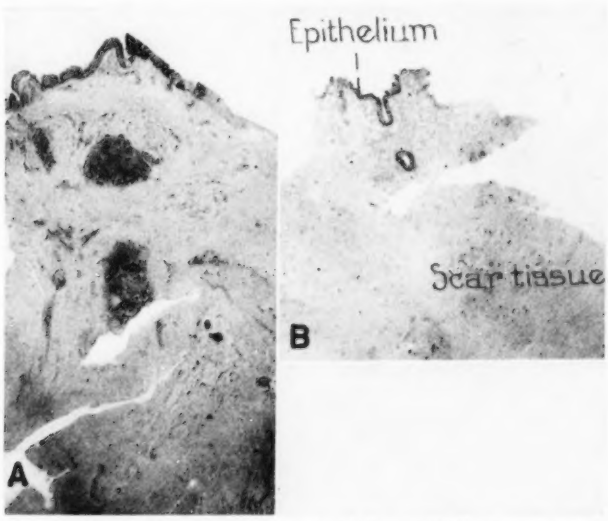

FIG. 31.—The epithelium in this case is of the stratified columnar variety. (A) The dark staining areas are accumulations of leukocytes in the scar tissue. (B) Branching gland duct (x200).

ated columnar pseudostratified epithelium resting on white fibrous scar tissue. No glands were present. In a small area such as this one the epithelium can return within a month. When about one third of the sinus lining was removed (in two cases) and two years later we reoperated and removed the entire lining, we found in the microscopic sections of the unoperated portions of the sinus membrane (1) a layer of pseudostratified ciliated columnar epithelium; (2) a superficial layer of areolar connective tissue; (3) mucous and serous

we make a counter opening (antrum window) in the nasoantral wall. A flap of nasal mucous membrane, several millimeters in width, then can be turned into the antrum to cover the floor of the window and antrum. At a later operation these tissues may be removed and examined histologically. If we do so, we will find that the nasal flap contains many mucous and serous glands and rests on scar tissue below. The scar tissue shows fibroblasts in the superficial portions and dense wavy collagen fibers deeper in the lining.

After complete removal of the mucous membrane, a layer of granulation tissue forms over the denuded bone. Within four weeks the epithelium of the nose begins to spread into the sinus, growing over the granulation tissue. The advancing border is composed of flattened cells, followed by cuboidal cells, which, in turn, are followed by columnar cells. At the end of four months, if all goes well, the entire cavity is epithelialized and covered by pseudostratified columnar epithelium. The mucoperiosteum of the sinuses aids in the pneumatization and formation of the cavities. After pneumatization is completed, it continues to act as a periosteum. It may participate in the proliferation of new bone or absorption of existing bone. Removal of the mucoperiosteum results in a proliferation of new bone, as seen in the sinuses that have had a radical exenteration of the lining. This proliferation also is seen in certain forms of chronic sinusitis, particularly purulent sinusitis. Since glands are not abundant, the epithelium of the sinuses must moisten the air in order to avoid desiccation of the surface. This function is not as great in the sinuses as it is in the nose. The mucoperiosteum of the sinuses acts as a covering in places where dehiscences are found in the bone. They thus participate in the general defenses of the body against infection and probably elaborate antibodies and participate in general immunity.

Although the scar tissue is better than a diseased and infected mucous membrane, it is a poor substitute for the real thing, and, like false teeth, has so many faults that I hesitate to call it a regeneration. When scar tissue and bone obliterate the sinus cavity, which sometimes is desirable, for the cavity itself has no function worthy of a name, the end result is not regeneration. In some cases, even after the most radical surgery, the disease continues and at a subsequent operation it will be found that the scar tissue itself contains implantation cysts and abscesses deep within its substance. These buried foci are apt to follow curettage and incomplete removal of infected bits of tissue, because epithelial cells are cohesive and have a tendency when implanted in granulation tissue to form implantation cysts.

PRINCIPLES OF NASAL AND SINUS SURGERY

From the foregoing, it is evident that a denuded area of bone in the paranasal sinuses will eventually cover with scar tissue and that this will heal over and become epithelialized. The new-formed scar tissue may be thick or thin, and there usually is a great deal of new bone deposited under it. The cavity, therefore, will cast a heavy x-ray shadow and subsequent examinations may lead to a diagnosis of sinusitis.

Any surgery that is attempted in the sinuses must, *first*, preserve the natural ostium or duct through which a sinus normally drains; this is especially true of the frontal duct, but also applies to the ostium of the maxillary and other sinuses. *Second*, windows and openings into the sinuses should be as far from the ostium as possible, and in the maxillary antrum the window is best made under the inferior meatus. *Third*, the interior of the sinus should not be curetted, and any mucous membrane that must be removed should be removed systematically and by subperiosteal dissection. Hilding's suggestion that we make the window opening in the bone a little larger than that in the mucous membrane, thus allowing the mucous membrane on either side to meet over the bone, is a good one, as is the technic for making a window in the sphenoid, which is quite similar, a suggestion that we owe to Proetz.

As regards the frontal sinus, the preservation of the epithelium of the duct is essential, but where radical surgery is necessary and the entire mucous membrane must be removed, it is well, whenever possible, to follow the technic developed by Sewall in which a flap is transplanted from the nasal mucosa into the frontal sinus, resting along the intersinus septum. In all sinus surgery, wherever the mucous membrane is removed, it should be removed carefully by subperiosteal dissection in order to prepare a smooth bed and prevent any remnants of epithelium from remaining behind. These epithelial remnants tend to produce implantation cysts. Small bits of epithelium that remain in scar tissue tend to grow toward each other and epithelium has the capacity to make a cyst when it is caught up in granulation or scar tissue.

And last, but not least, a nasal synechia is a fibrous band between two opposing areas of mucosa, due to disease, wounds, postoperative adhesions, or cauterizations. The granulations of two denuded or ulcerated areas unite to form a fibrous bridge across the nasal cavity, which later becomes epithelialized scar tissue.

FIG. 32.—(A) Attachment of the middle turbinate. The two ethmoid sinuses are shown on the left and below them the thickened lining of the bulla ethmoidalis. Note the difference in thickness and vascularity of the turbinal tissues. (B) Concha bullosa (cystic middle turbinate), showing an ethmoid sinus within the middle turbinate (x10).

CHRONIC RHINITIS

The nasal turbinates act as radiators of heat and moisture, and cannot be destroyed with impunity, as was done a generation ago. Though the middle turbinate may interfere with drainage through the middle meatus or be impacted against the nasal septum, which frequently produces headache, the disorder often can be corrected by fracturing the turbinate in the required direction. This minor operation does not harm the physiology of the nose, and may do it some good.

Concha bullosa may require resection of the turbinate to relieve a chronic headache, which is best done by splitting the turbinate from front to back, thereby preserving the septal (convex) half in which the olfactory epithelium is present.

CONCHA BULLOSA

Concha bullosa is the name given to an enlarged middle turbinate in which

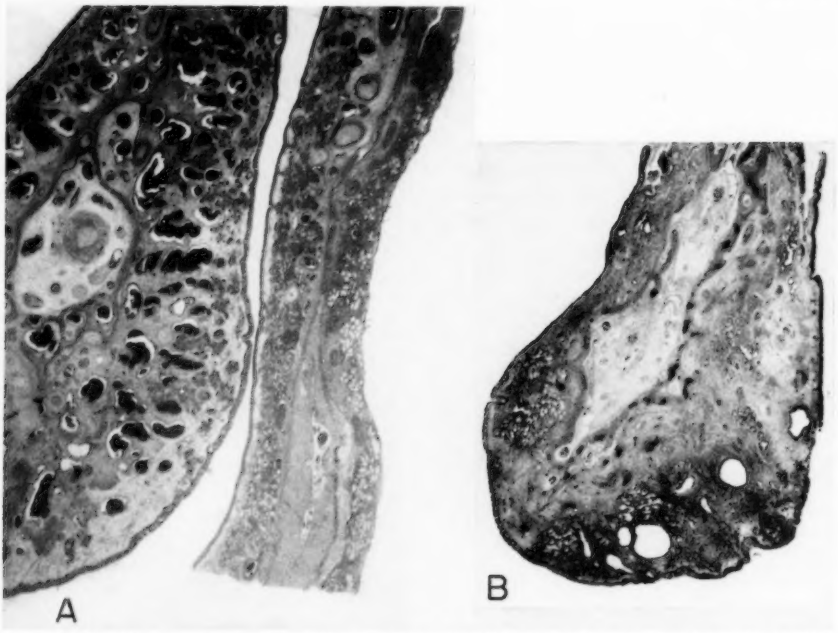

FIG. 33.—(A) Normal nasal turbinate and septum. (B) Atrophic turbinate from a case of atrophic rhinitis. Note the difference in size and vascularity.

an aberrant ethmoidal cell is found. This anatomic condition is sometimes called a cystic middle turbinate, and it is seen in about 10 per cent of women, producing an unusually large and bulky turbinate, obstruction of the nose and a contact or pressure headache.

When palpated with a probe, there is a definite sensation of hollow bone. The outer or convex aspect is covered with nasal mucous membrane while the interior is lined with a thin mucous membrane exactly like that of the ethmoidal sinuses.

Since the ethmoidal cell lies within the middle turbinate, a sinusitis may develop here, and depending upon the type of disease, it may be an empyema or a hyperplastic sinusitis. In most instances, however, the aberrant cell is perfectly normal (fig. 32).

OZENA

Ozena, a distinct disease entity, is characterized by rarefaction of the turbinates and atrophy of the mucous membranes. It affects women more often than men and produces a putrid discharge, extensive crust formation, abnormal patency of the nasal passages, anosmia, sometimes saddle deformity of the nose and pharyngitis sicca.

Though sinusitis is common in this peculiar form of atrophic rhinitis, it apparently is a result rather than a cause of the disease. In some instances the sinuses are normal in appearance. In one case that I have been treating for many years, I found eosinophils in the discharge.

In children it begins as a mucopurulent rhinitis with crusting during adolescence and atrophy in adult life. Prob-

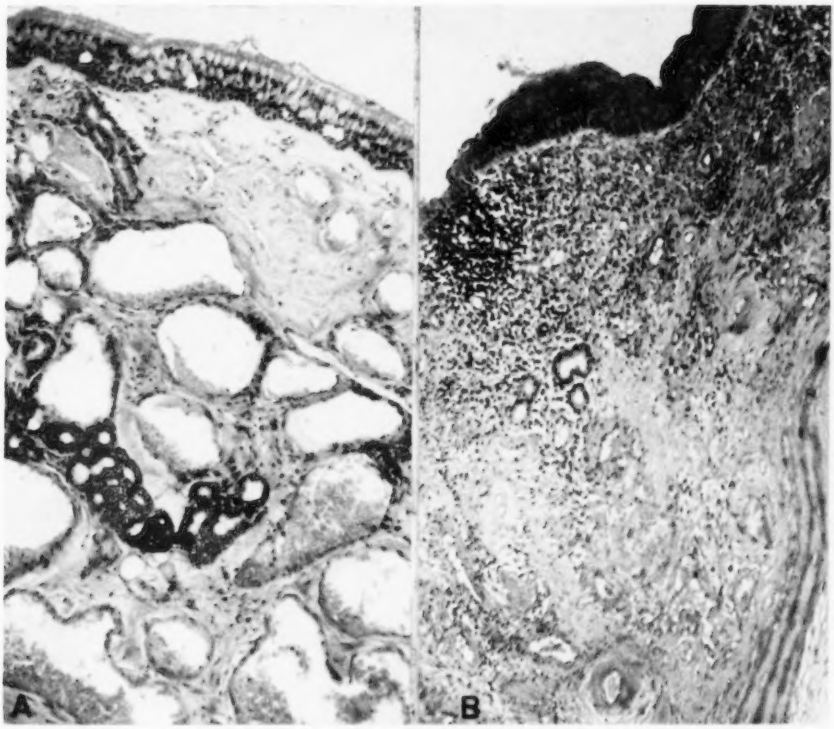

FIG. 34.—(A) Normal nasal mucous membrane. The epithelium is pseudostratified and ciliated. The empty spaces are corpora cavernosa (erectile tissue). Most of the blood cells have been washed from the blood vessels. Note the glands and absence of cellular infiltration in the subepithelial connective tissue. (B) Atrophic rhinitis. The epithelium is metaplastic and of the stratified squamous type. Note the loss of cilia. There is a dense infiltration of inflammatory cells in the subepithelial connective tissue. The blood vessels and glands have practically disappeared and are atrophic. Compare A and B. Both have been photographed with exactly the same magnification.

ably of constitutional origin, it appears to be an endocrine disorder (fig. 33).

The inferior turbinate undergoes atrophy in form and function and in the advanced stages of the disease it is much smaller than normal, owing to a striking reduction in the amount of bone and mucous membrane. The epithelium shows marked metaplasia to the stratified squamous type. The submucosa is fibrotic, poor in vascularity and glands and is much reduced in volume. Inflammatory activity is revealed by infiltration of any round cells, lymphocytes, plasma cells, hyalin bodies and pyogenic leukocytes. An important feature is the obliterative arteritis in the deep arteries, which reduces the vascular channels to mere slits. Sclerosis and atrophy of the blood supply and mucus-forming glands interfere with the warming and moistening of the inspired air, creating excessive drying, stagnation of discharge and crust formation within the nose. The nasal mucosa is not as sensitive as normal (fig. 34).

RHINOSCLEROMA

Clinically, rhinoscleroma is sometimes confused with atrophic rhinitis. In both

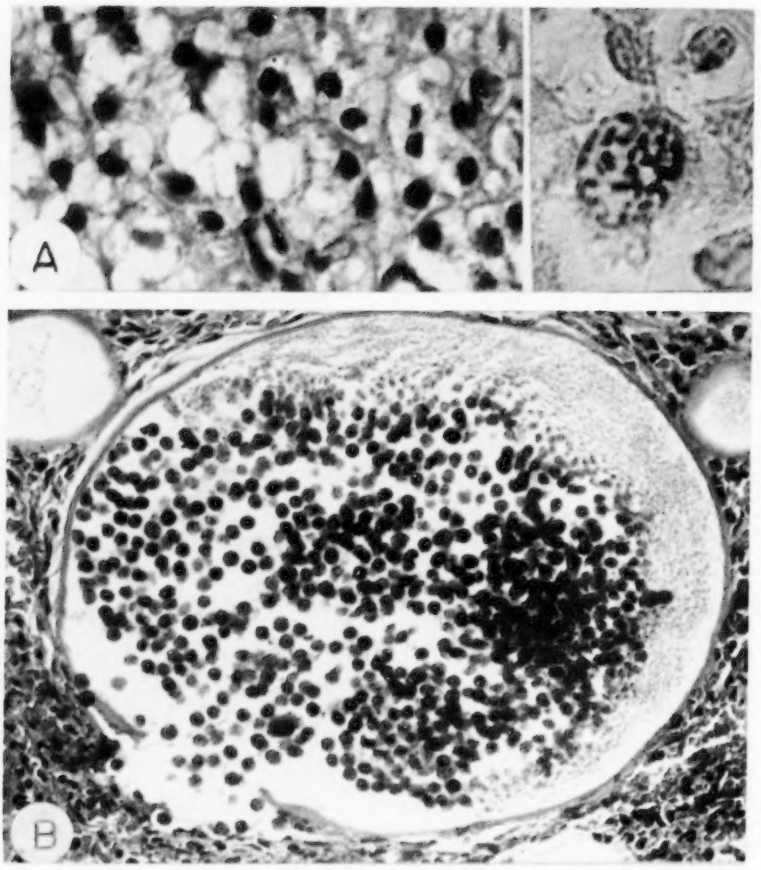

FIG. 35—(A) Scleroma showing the macrophages, or foam cells, and bacilli. (AFIP 65746) (B) Rhinosporidiosis. A cyst filled with the spores. (AFIP 82097)

conditions there is a foul-smelling purulent nasal discharge with much crusting.

Scleroma is a chronic granulomatous disease that attacks the mucous membranes, but not the bones and cartilages, of the nose, pharynx and other portions of the respiratory tract. It is readily recognized in its more acute stages, producing nodular lesions that are soft and granulomatous, while those of longer standing become hard, producing atrophy and deformity.

Histologically, the picture is that of a granuloma. There is perivascular infiltration in which the Mikulicz cells (large vacuolated cells with a small round nucleus) contain the bacillus, Klebsiella rhinoscleromatis, which is considered to be the specific etiologic cause of the disorder. Russell's bodies (modified plasma cells) are also seen. (The bacillus [Gram-negative] that causes the disease responds to streptomycin.) There is a marked proliferation of dense col-

lagenous tissue that is transformed into cicatricial tissue.

In the late stages, the fibrotic lesions differentiate this disease from other granulomatous diseases. The atrophic and fibrotic processes follow the granulation tissue. The granuloma is composed of young blood vessels, lymphocytes, plasma cells and large mononuclear cells with a rather foamy appearance. The fibrotic stage or scleroma exhibits irregular bands of connective tissue containing considerable collagen and a large amount of scar tissue.

RHINOSPORIDIOSIS

Rhinosporidiosis is due to a fungous infection. It usually is seen in males and is characterized by a polypoid granuloma that develops on the nasal septum. The typical polyp of rhinosporidiosis usually occurs singly, although one occasionally may encounter two or more such polyps. Grossly, they appear reddened and pebbled like a raspberry.

Histopathology. The histopathology consists of a fibrous polyp with a foreign body granuloma reaction and occasional tubercles. The epithelium covering the polyp may be of the stratified squamous type. The underlying connective tissue is very vascular.

A striking feature about rhinosporidiosis, however, is the formation of many perfectly round cystic cavities in the polyp, which contain the spores. These cysts may contain as many as 16,000 spores when ripe (fig. 35B). The spores have a large nucleus and a small rim of cytoplasm. The young spores are small and dark, and solid nuclei appear in them. To the naked eye, the sporocysts appear as small yellowish spots. Treatment is by wide and deep excision.

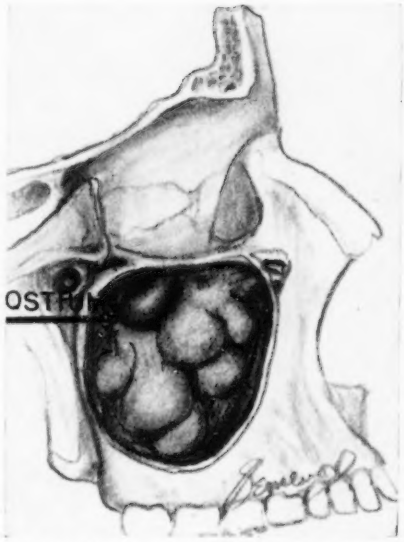

FIG. 36.—Mucous retention cysts of the antrum.

CYSTS OF MAXILLARY SINUS

Four *types* of cysts are found in the antrum: (1) mucous gland retention cysts; (2) serous cysts; (3) dental root or radicular cysts; and (4) dentigerous or follicular cysts. In discussing antral cysts, we must consider two totally different categories.

The first category includes the cysts that arise within the mucous membrane of the sinus. The cysts, as has been stated previously, are of two types: (1) mucous gland retention cysts (fig. 36), and (2) serous (nonsecreting) cysts (fig. 37).

Mucous Membrane Origin

1. The *mucous gland cysts* are due to obstruction of glands. They contain mucus and usually are small and multiple, but they may become large and lie along the floor of the antrum. They usually arise in the medial half of the sinus.

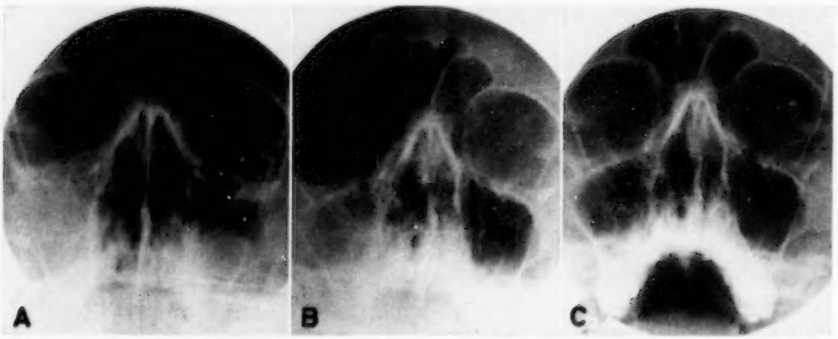

FIG. 37—Serous cysts of the antrum. (A) This patient has a large cyst in the right antrum which is completely opaque, and a smaller one in the left antrum. (B) Another patient showing opacity of the right antrum due to a large serous cyst. (C) Postoperative x-ray of the same patient. Many of these cysts respond to simple conservative surgery—rupture of cyst through an antrum window.

2. The *serous cysts* arise within the sinus mucosa. They are devoid of epithelial lining and are merely pockets of tissue or lymphatic fluid. The recovery of yellow-brown, clear fluid during antrum puncture suggests a serous cyst. It clots on standing. Cysts arising from the jaws or teeth, include the radicular cysts and the follicular cysts, both of which are periodontal in location.

Dental Origin

Dental root cyst (periodontal cyst, radicular cyst, periapical or lateral cyst). This cyst is lined with flattened squamous epithelium, sometimes low cuboidal and occasionally columnar epithelium. It usually forms at the root end of an infected tooth, *the roots* of which project into the cyst cavity. Such teeth may have been extracted while the cyst remains (residual cyst), or the cyst may lie lateral to a tooth. It arises from a chronic granuloma or "root abscess," and the epithelial lining is derived from the periodontal membrane which surrounds the dental follicle. The capsule is connected to the periodontal (peridental) membrane and is infiltrated with chronic inflammatory cells. The cyst contains a yellow-brown, clear, syrupy liquid, in which cellular debris and cholesterol crystals may appear. The wall of the cyst, which protrudes into the antrum cavity when it is large, is formed of fibrous tissue.

Dental root cysts are due to a granuloma or chronic inflammation of the periosteum surrounding the root of the tooth. Composed of compact connective tissue, the sac is lined with epithelium and infiltrated with chronic inflammatory cells. The cyst may elevate the floor of the antrum or bulge the lateral nasal wall. The cysts are subdivided into (a) the apex type, (b) the lateral type, and (c) the residual type.

Dentigerous cysts (dentigerous or follicular cyst or eruption cyst). This type is lined with stratified squamous epithelium. It contains a viscid straw-colored fluid and usually one or more imperfectly developed supernumerary teeth. The dentigerous cyst is of developmental origin, and is due to proliferation of the cells of the enamel organ of an unerupted tooth (partially or completely formed), the crown of which lies within the cyst. The dentigerous cyst is surrounded by a parchment-like fibrous capsule or bony shell, which may be thin or thick, and lies about the unerupted tooth, usually the third molar. The

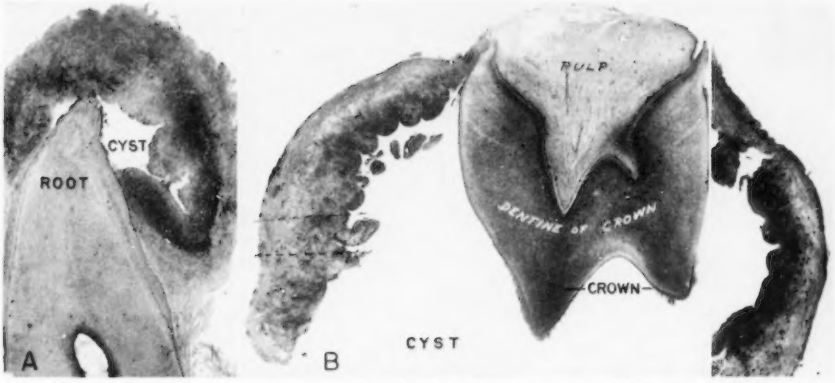

FIG. 38. (A) Root abscess or apical granuloma of a beginning root cyst. (AFIP 54202) (B) Dentigerous cyst. The crown of the tooth lies within the cyst. (AFIP 60393)

capsule shows chronic inflammatory cells. The follicular cyst is similar to a dentigerous cyst but exists without an embedded tooth. These cysts may be very large. The entire lining must be removed. The sac contains the crown of an isolated tooth and has a thick fibrous wall surrounded by a thin bone. Dentigerous cysts may become very large and completely fill the maxillary antrum.

The *developmental cysts* of the maxilla include: (1) median cysts (median palatine cysts), (2) incisive canal cysts, (3) globulomaxillary cysts and (4) an odontogenic neoplasm of a cystic nature, the ameloblastoma.

DEVELOPMENTAL CYSTS

Nasoalveolar cysts appear in the lateral nasolabial region, the lateral wall of the nasal vestibule. The cysts have a fibrous capsule and columnar, cuboidal, ciliated or stratified squamous epithelium.

Globulomaxillary cysts occur between the roots of the lateral incisors and canine teeth (the junction of the globular and maxillary processes).

Nasopalatine cysts are found in the region of the incisive canal or foramen. These cysts may be very small and the epithelial lining may be either ciliated columnar or squamous in type. The ciliated cells are derived from the epithelial cord that extends in from the nasal cavity; the squamous epithelium comes from the oral cavity.

Median cysts (fissural midline cysts) of the maxilla are (1) the *median alveolar cysts* that occur between the roots of the incisor teeth and have an elliptical shape, and (2) the *median palatal cysts* that are found farther back on the hard palate and are lined by squamous epithelium.

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A QUANTITATIVE AUDIOLOGIC TEST

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SALINA, KAN.

IT IS my intention to introduce a new test to be used in cases of defective hearing or partial deafness due to allergy, a quantitative audiologic test. This procedure is an extension of Dr. Hugh A. Kuhn's test whereby an audiogram of the patient is taken, a dose of medication is administered, and after a suitable waiting period a second audiogram is made and compared with the first. The resulting difference is interpreted as being favorable or unfavorable as to prognosis.

To avoid the errors of inconsistency which occur when a patient takes a medication orally, it was decided to use an injectable antihistamine; this choice was made because many cases of allergy will improve temporarily under the use of an antihistamine. However, this test can be performed with any antihistamine, oral or injectable, or cortisone, or ACTH. The drug used in this test is chlorprophenpyridamine maleate solution 100 milligrams per cubic centimeter, which is marketed under the name of Injectable Chlor-Trimeton® Maleate.*

To perform the test, a careful initial audiogram is taken and the patient is given Injectable Chlor-Trimeton® Maleate hypodermically from a starting dose of 2 milligrams. The dose is increased by 2 milligrams at two-day intervals until the maximum improvement in hearing is definitely obtained, or until it is certain that a patient who should be receiving histamine is being given an antihistaminic, or until, in some cases, drowsiness is produced. If drowsiness occurs, the drug may be continued at that dosage level unless the symptom is objectionable to the patient, in which case the dose may be lowered 2 milligrams and the test continued until the maximum gain is apparently obtained. During this waiting period, a check audiogram is taken once a week to observe the rate of change, and when it becomes apparent that no further gain may be obtained, a final audiogram is taken and compared with the initial audiogram and the drug is discontinued. The difference between the initial and the final audiograms is the estimated gain in hearing to be expected for that patient under proper allergic management (figs. 1 to 3).

The advantages of using this method of audiologic testing follow:

1. An estimate of the gain in hearing to be obtained by proper allergic treatment is possible to both the doctor and his patient.

2. Cases which show a satisfactory estimated gain by this method will respond to proper allergic therapy and management.

3. It does not depend upon the patient's ability to follow orders over a period of time.

4. This test gives the patient confidence in both his doctor and the allergic method of treatment.

5. Injectable Chlor-Trimeton® Maleate is painless and well tolerated.

Presented at the meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Oct. 17, 1952, Chicago, Ill.

*This drug was obtained through the courtesy of Dr. George Babcock, Jr., of the Research Division of Schering Corporation.

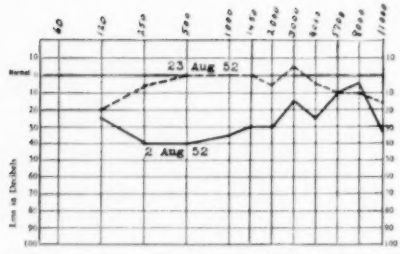

FIG. 1.—A 26 year old white female had progressive defective hearing for two years. This audiogram shows the amount of improvement from August 2, 1952, to August 23, 1952, with CTM 100.

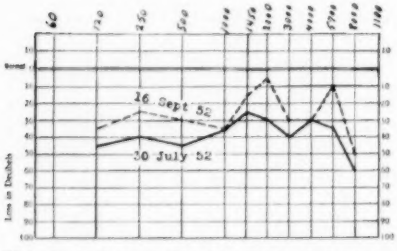

FIG. 2.—A 49 year old white female had bilateral chronic otitis media and defective hearing all her life. The gain in this audiogram was made from July 30, 1952, to September 16, 1952, with CTM 100.

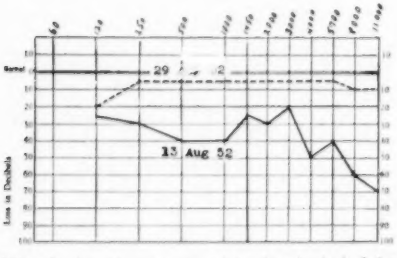

FIG. 3.—An 18 year old white female had defective hearing of the right ear for three years. Gain in this audiogram was made from August 13, 1952, to August 29, 1952, with CTM 100.

6. Cases that should be treated by histamine can be discovered by this procedure and therapy instituted.*

7. Intercurrent attacks of common cold merely delay the conclusion of the test if the drug is continued through the period of infection.

The disadvantages seem to be limited. (1) Patients requiring histamine therapy become worse. (2) Rare attacks of epistaxis occur which almost never recur if the current dose of the drug is suspended for a week or more and the same dose of the drug is resumed. Those cases of epistaxis which did occur were associated more with weather changes than with drug therapy. (3) The maximum dose of the drug varies with each patient. Well-tolerated doses range between 4 and 16 milligrams.

CONCLUSION

This test represents a quantitative audiologic examination which seems to be useful in estimating the degree of defective hearing due to allergy.

I do not know whether this test would be applicable to anything other than allergic deafness. It was devised as a method for seeking out cases of deafness due to allergy, and only deafness due to allergy.

We have cases of otosclerosis which did not improve under this drug and which showed no gain whatsoever. We have some cases of auditory neuritis which showed absolutely no improvement. In my estimation, this test is strictly for a case of deafness due to allergy.

Deafness due to allergy is any form of deafness in which we find that pathology due to allergy, whether it is otitis media on a serous basis, whether it is eustachian salpingitis, whether it is otitis externa, with the production of a huge amount of debris blocking the acoustic apparatus, or whether it is something in the cochlea, say, a hydrops. Any of these can be due to allergy, and too many times they are due to allergy and neglected.

* Cases of defective hearing requiring the use of histamine very frequently appear as cases of allergy with deafness. If there is no response to the use of this drug, or hearing evidently becomes worse, it is to be presumed that a trial of histamine is indicated in such cases.

THE COMMON GROUND OF ALLERGY, AUTONOMIC DYSFUNCTION AND ENDOCRINE IMBALANCE

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ST. PAUL, MINN.

MAY I borrow the gracious habit of another generation and offer first an apology for this presentation? I labor under no illusions that I present something new or strikingly original. Rather it is intended to offer a perspective—a viewpoint which can make the approach to some of our common clinical problems simpler; a viewpoint correlated with the ultimate goal of intelligent therapy; a viewpoint which has at least the merit of integrating current attitudes stoutly defended each by its individual proponent. There is danger in oversimplification. But this danger should not discourage one from efforts to reduce problems to basic terms.

Medical specialism can be viewed from two standpoints. The one embraces familiarity with a restricted segment of the body and the influence of the functions and disorders of that segment upon the organism as a whole. The other perspective views the whole organism and the influence of its functions and disorders upon the restricted segment. The future and usefulness of medical specialism depends upon the willingness of the specialist to accept the latter viewpoint. Medical methods which dismember the patient can only establish a bizarre hodge-podge of medical knowledge which, from divergent angles, resembles a cubist's nightmare rather than an orderly system of facts and theories related to the whole human being. Witness, for example:

If a young woman in the first trimester of pregnancy describes a recently acquired and persistent nasal obstruction and mucoid nuisance to her obstetrician, he recognizes it as a not uncommon endocrine imbalance which, if untreated, may improve as the pregnancy proceeds or may persist until the early postpartum period. A psychiatrist, on the other hand, might be pardoned for suspecting an emotional conflict, perhaps allied to her pregnant state, as the source of her nasal problem. The rhinologist versed in nasal allergic problems and their management might well indict the dust inhalant features of the rug and pad in the new living location. It is obvious that this young woman has a definite etiology for her individual problem which is not going to vary with the clinical threshold she crosses. Her differential requires a broad view of her as a total being and not a package of parts.

Allergic reactions, states of autonomic dysfunction and of endocrine imbalance can produce identical clinical symptoms. In the field of otolaryngology they are commonly expressed as rhinitis, vascular pain, myalgia (cervical or pharyngeal) or inner ear disturbance with variable degrees of tinnitus, hearing impairment and vertigo. The common denominator in this wide range of expression is abnormal extravascular fluid. It creates the immediate problem in the end organ. The method of its formation in the precipitor states of allergy, autonomic dysfunction and endocrine imbalance may vary but the end result in local symptoms is the same

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when excessive extravascular fluid is established in the end organ tissue.

Of all the peripheral tissues involved in extravascular fluid imbalances, these of otolaryngology, as noted above, are among the most frequent. Therefore, the otolaryngologist must equip himself with the differential knowledge to seek the specific cause in the individual case, or he must equip himself with the knowledge that a differential exists and someone qualified must seek it out. Results are not obtained by a rigid approach to the problems of transudate through the narrow and limited view of allergy alone, or of endocrinology alone, or of psychosomatic concepts alone.

It is the purpose here consciously to avoid exhaustive pro and con bibliographic references to the mechanisms of transudate formation in these three states—allergy, autonomic dysfunction and endocrine imbalance. There is much that is known of these mechanisms and much more that is not. The purpose of this discussion is better served by concise reference to accepted fact and theory.

The state of altered reactivity or allergy in a confined sense implies the presence in the shock organ of the allergic individual of antibodies in intimate association with smooth muscles cells—principally of the arteriolar bed. The antibodies are an immunologic response to antigenic stimulation. There is evidence that this immune reactivity is mediated through the basal nuclei of the brain—principally aggregated in the hypothalamic region. The antibody, once established in association with the smooth muscle, stimulates the latter to spasm when the organism is re-exposed to the originating antigen. Possibly from the resultant ischemic hypoxia and starvation of its endothelium, the capillary tube becomes abnormally permeable and fluid exudes into the extravascular spaces. This sequence is widely accepted as the method of transudate formation in the allergic reaction.

Autonomic balance is mediated through the basal nuclear aggregates of the brain stem. In this central switchboard the neurovascular, neuromuscular and neurosecretory activities of the vegetative nervous system are integrated. Reflexes from body surface receptors stimulated by heat, cold, light, barometric variations, etc., and impulses from higher psychomotor areas play through these centers upon the autonomic activity of peripheral organs. Unbalanced expression in peripheral organs results when integration is faulty. Faulty neurovascular impulsion has its principal influence on the arteriolar bed. Here sphincteric spasm indistinguishable from the antigen-antibody conflict of the narrow allergy can result in similiar damage to the capillary endothelium and capillary leak. The resulting transudate problem is indistinguishable from that of the narrow allergy.

Endocrine imbalance has a less well-defined mechanism of transudate. The thyroid, the gonadal and the adrenal cortical hormones all are important to the proper position of body fluids. In endocrine variations from the normal the three possible levels of endocrine fault must be considered. Of the first or primary mediator of the pituitary little is known. It is considered to be a direct or indirect stimulus pathway from the basal nuclei to the anterior lobe of the pituitary. The second level is the trophic hormone level of the pituitary itself. And the third level is the level of the target gland—principally, in fluid balance considerations, the thyroid, the gonads and the adrenal cortex. Outspoken, gross signs of endocrine imbalance are the exception, not the rule. Evidence of endocrine imbalance must be sought for. Less-than-myxedema grades of hypothyroidism present no standard complex of clinical

signs. A transudate problem warrants thought of thyroid deficiency as a possible cause. The concurrence of transudate problems and the menopause or male climacteric is the best evidence of deficient estrogen or testosterone source. The cyclic premenstrual accumulation of extravascular fluid with the premenstrual symptoms of headache, bloating, endolymphatic hypertension or pharyngeal myalgia is the best evidence of the relation of high estrogen levels to the phenomena. The collagen diseases so intimately associated with adrenal cortical dysfunction are transudate disorders first and for a long time before collagen formation. Therapy is of little value in the collagen phase. These many abnormal endocrine-caused shifts in fluid balance may result through one or several mechanisms. Little is known of them. They may include changes in the capillary endothelium itself, shifts in electrolyte balance between vascular and extravascular spaces, and tissue change such as the increase in the subcutaneous mucopolysaccharides of myxedema. Whatever the mechanism, transudate is the end product.

The patient with an abnormal extravascular fluid problem above the clavicle and excluding the eye and the brain is prone to gravitate to the otolaryngologist. The clinical syndromes of rhinitis, myalgia, vascular pain and labyrinthine ischemia and transudate are not difficult to recognize if one is willing to honor suppuration only when it truly exists and to cast out vague references to meaningless atypical neuralgias and pigeon-hole headaches. Once the presence of a transudate syndrome is established, it is up to the otolaryngologist or his reference physician to determine the relative responsibility of allergy, autonomic dysfunction or endocrine imbalance. The problem is not made easier by the possibility of an interplay of all three causes. This is particularly apparent when it is noted that all three are related directly or indirectly to the mediating influences of the basal nuclear aggregates. Faulty function in this central area may be due to the inherent inadequacies of a poor family brain stem or the acquired inadequacies of a postpoliomyelitic, postencephalitic or postarteriosclerotic basal nuclear change. It is conceivable that this faulty function can be evidenced through all three transudate sources—the immunologic mediation of allergy, autonomic impulsion directly, or the primary mediation directed from the brain stem to the trophic producing pituitary—all in the same individual. Thus, a youthful hypothyroid can also manifest specific inhalant or food sensitivity. Desensitization or elimination is only partially corrective. Addition of a proper amount of thyroid substance can make him symptom-free. Thus, a woman at the menopause, and in the absence of extraovarian estrogen sources to tide her through the critical period, may the more readily develop a transudate problem in her inner ear under the stress of physical exposure or severe emotional strain. Thus, a recurrent vascular head pain occurring cyclically in the immediate premenstrual period and additionally, on occasion, after ingestion of a particular food, or exposure to an annoying draft, or an emotional upset is a clinical commonplace. Thus, the sudden onset of acoustic and vestibular symptoms usual with an abrupt increase in endolymphatic fluid pressure may be attributed by the victim to a stressing emotional upset, severe chilling of the body through physical exposure, or the gluttonous ingestion of a dozen hard-boiled eggs of which he is fond in spite of previous unfortunate experiences with them. Any or all of these factors might well be only the final transudate straw in the victim who was unknowingly significantly hypothyroid. It would be

an interesting study to call back a large labyrinthotomy series to check the basal metabolic rates and blood cholesterols. And hypothyroidism is only a single unbalanced endocrine substrate possibility.

Is it enough then to treat the results of transudation in terms of vascular pain, rhinitis, myalgia or inner ear syndromes in a standard, predetermined manner with hormones or dehydration therapy, with elimination diet or histamine, with pharmaceuticals or psychotherapy? One suspects not. Better results can more reasonably be expected by a sensible correction of all the causative factors in the individual case than by the stereotyped approach indicated by the sign on one's door.

One of the universal truths about transudate problems is this. The peripheral expression, whether nasal, acoustic, vestibular or painful, tends to recur and produces sufficient distress to add fatigue and emotional stress to the problem as a result instead of a cause. An understanding doctor with human insight is an excellent psychotherapist. If he can bring to the problem in addition, a knowledge of specific reactivity, endocrine interrelationship and the pharmacy of stress, he is ideally suited for the work. Above the clavicle the man with whom the problem is first placed is commonly the otolaryngologist. He is equipped to examine the respiratory tract, to evaluate the ear, to rule out suppurative source, and in all ways, to complete the examination of the transudate problem. He may content himself with identification. If not, he must then equip himself with the differential of transudate — to correctly balance the etiologic possibilities in every case between specific allergy, autonomic dysfunction, or endocrine imbalance. The aim of medical progress is, after all, a more satisfactory result for the patient even though it batters down the protective ramparts of narrow specialism.

SUMMARY

1. The common denominator of allergy, autonomic dysfunction and endocrine imbalance is transudate formation.

2. In the tissues of the ear, nose and throat the transudate most frequently expresses itself as rhinitis, myalgia, vascular head and neck pain and variable combinations of inner ear symptomatology.

3. The symptoms are the same regardless of the source of transudate.

4. The specific mechanism—allergy, autonomic dysfunction or endocrine imbalance—in the individual case must be identified as a prerequisite to reasonable therapy.

5. Frequently, more than one of these mechanisms is related to a single case.

VASCULAR HEADACHES AND RELATED PHENOMENA

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IN THE preallergic, preantibiotic and prehistamine days of otolaryngology, the management of practically all conditions in this field was approached primarily from the surgical standpoint. So the otolaryngologist was primarily a surgeon, and in spite of the more recent modern developments in therapy he is still rather reluctant to admit that his specialty has become predominantly medical. Far too few have taken advantage of this fact and consequently are overlooking opportunities to render a better and more satisfactory service to the patient. It is evident that the plan of graduate teaching in otolaryngology must be modified to conform to both the medical and surgical problems as they are encountered in practice today, not yesterday or yesteryear. Eventually these changes will gradually come about as the result of the proper education of the younger men and the beginners in otolaryngology.

So in the "Neanderthal" days of otolaryngology, the cause of headaches and various related pains in the face, the eye, various parts of the cranium and the neck was directed to sinus disease, chiefly the hyperplastic type, septal spurs and deviations, or to involvement of the nasal ganglion and its branches as the result of neuritis or adjacent sinusitis. Now we recognize hyperplastic sinus disease as an allergic phenomenon and the nasal ganglion syndromes as chiefly of vascular origin. All types of organic pathology, however, must be considered in the diagnostic investigation. On the other hand, certain associated pathologic conditions, such as a spur on the septum, should not be considered as very likely causes of headaches. The following conditions may or may not be related to the problem under consideration.

Nasal and sinus allergy; polyps, tumors and cysts; acute, subacute or chronic infection; dental infections; temporomandibular joint syndrome; deflections and spurs of the septum; tonsils, adenoids, lingual tonsil — tumors; elongated styloid process; head trauma; eye conditions, such as, scotomas, retrobulbar neuritis, glaucoma, uveitis; cranial and intracranial lesions; temporal arteritis; cervical myositis; hypoglycemia; endocrine disorders; psychogenic factors; syphilis.

With these various conditions, pain is usually associated with direct nerve involvement as a neuritis or the result of some form of pressure, such as that from retained secretion in the sinus or from intracranial lesions. Posttraumatic headache is not infrequently of the vascular type and responds to adequate histamine therapy. Some types of associated eye disease may be of vascular origin. Cervical pain may be vascular or muscular.

NEURITIS AND NEURALGIA

In the conditions of neuritis and neuralgia, the pain follows the course of the anatomic distribution of the nerve or nerves involved. With neuritis the pain is more or less constant, at times sharp, but usually aching in character. Paresthesia may also be present. Inflammation, pressure or tumor involvement

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is usually the cause. True neuralgias, such as those of the trigeminal and glossopharyngeal types, represent typical syndromes characterized by the occurrence of sharp paroxysmal pain which follows the exact distribution of the branches of the nerves involved and which is associated with triggers.

INVOLUNTARY NERVOUS SYSTEM PHENOMENA

The following phenomena may be associated with headaches of vascular origin: (1) secretory — lacrimal, nasal, salivary; (2) hyperhidrosis—sweating; (3) thermal—increased temperature.

VASCULAR PHENOMENA

The various types of patterns of vascular headache and related phenomena encountered are dependent upon one or more of these basic factors: (1) vasoconstriction, (2) vasodilatation, (3) spasm and (4) edema. The edema phase of headache may be associated with migraine but is most important in allergic headache.

The high incidence of vascular phenomena involving the structures of the head and neck warrants greater attention to these problems on the part of the otolaryngologist. It is especially important to recognize some of the unusual complaints which are referred to the pharynx, larynx, tongue, jaws, face, parotid region, or deep muscles of the neck. Upon physical examination, no pathologic condition can be found to account for many of the complaints of pain, sense of pressure, or foreign body. Because of this fact we used to classify patients with such complaints as neurotics. The symptoms could not be associated with any particular structure or nerve. These phenomena can now be classified as vascular disturbances, and they respond promptly to histamine therapy. The symptoms can almost always be assigned to certain arterial distributions.

HEADACHE AND RELATED PHENOMENA

Because of the high incidence of vascular phenomena in the field of otolaryngology and the development of successful methods of treatment, this phase of medical otolaryngology, in view of what has been lost from surgical procedures, should be a most welcome addition to our economic situation.

Although we are primarily interested in the headache problem, it has been discovered that other conditions may be classified as of vascular origin.

The entire group may be listed as follows: migraine; atypical migraine (equivalents); tension headache; histaminic cephalalgia; atypical histaminic cephalalgia; posttraumatic headache; auditory phenomena—vertigo, tinnitus, deafness; facial paralysis (Bell's palsy); ocular phenomena — ophthalmoplegic migraine, blindness, scotomas, retrobulbar neuritis; miscellaneous—facial pain, toothache, otalgia, parotid and temporomandibular joint pain, laryngeal and pharyngeal pain and discomfort, cervical pain and muscular discomfort; general — abdominal pain, precordial pain, pain in the extremities, multiple sclerosis, arthritis.

In our experience typical and atypical histaminic cephalalgia constitute the largest group of cases. More recently we have noted an increase in the incidence of the miscellaneous group because more attention has been directed to it. It is well to consider that these vascular phenomena may occur in any artery in the body. We have noted, not infrequently, that patients with headache and related phenomena have complaints of a vascular nature referred to the arms and legs. These symptoms were noted to disappear when the headaches were relieved.

A review of the literature on the subject of headache reveals a lack of classification and exact determination of the etiologic factors. Some observers em-

phasize the importance of food allergy, others classify most headaches as migraine. A commercial pamphlet recently published the following statistics from thirteen observers.

	PATIENTS	MIGRAINE-TENSION	HISTAMINIC CEPHALALGIA	OTHER TYPES
Otolaryngologists (3)	701	234	272	195
Internists and neuropsychiatrists (10)	710	637	18	55
	1,411	871	290	250

On the basis of these statistics it is evident that the incidence of migraine is far too high. The following studies indicate a much greater variety of types and causes.

Among 508 normal individuals, Vaughan[12] noted an incidence of headache caused by foods of 7 per cent. Among 247 patients with headache, Rowe[9] reported good results from food avoidance in 63.5 per cent. Although food allergy as a cause of headache may be overrated by some observers, it is frequently overlooked.

In recent studies conducted by Ogden,[8] it was noted that among 252 patients with nasal allergy, 58 per cent complained of frontal headache. Among 100 normals, the incidence was 27 per cent. He also reported that frontal headaches are usually bilateral and not infrequently the pain radiates to the temporal, vertex and occipital regions. In frontal headache, the anterior meningeal vessels are apparently involved (fig. 3).

In the review of a group of 4,634 individuals, Ogden found that 64.8 per cent had headaches, 47.1 per cent of which were frontal and 3.3 per cent were migraine. In the group of 2,184 patients with frontal headache, 46.7 per cent had nasal symptoms and 39.3 per cent of the latter had disturbances of vision with the headache. Twenty-five and one half per cent had definite eye symptoms.

In the total number of individuals in the survey conducted by Ogden (4,634), the various types of headache were found in the following percentages: migraine (broadly defined), 8.6; migraine (rigidly defined), 3.3; histaminic cephalalgia, 0.1; frontal headache, 47.1; and occipital or neck pain, 10.1.

Migraine

Wolff,[13] Friedman,[1] Friedman and von Storch[2] and Horton[6] define the syndrome of migraine as characterized by the following five factors: (1) periodicity, (2) cephalalgia, (3) gastrointestinal dysfunction, (4) cortical disturbance, (5) a familial history of migraine.

Atypical migraine. Periodic cephalalgia associated with any one of the latter three factors listed above.

Histaminic Cephalalgia

According to Horton[6]:

> Histaminic cephalgia is characterized by a unilateral headache which usually begins in the latter decades of life, is of short duration as it generally lasts less than an hour, commences and often terminates suddenly, tends to awaken the patient at night one to two hours after he has gone to sleep and is frequently eased by the patient sitting up or standing erect. It is associated with profuse watering and congestion of the eye, rhinorrhea or stuffiness of the nostril, increased surface temperature and, often, swelling of the temporal vessels on the involved side of the head.
>
> Pain is the outstanding complaint. It is constant, excruciating, burning and boring; it involves the eye, the temple, the neck and often the face.

Atypical histaminic cephalalgia. We[4] have also observed a large group of cases, somewhat more common than the

typical ones, which present most, but not all, of the characteristics of the typical type. They may lack the sweating and the tearing phenomena, may be of longer duration, may not begin or end suddenly, or deviate from the typical in some other features. As a rule, the pain is not as severe, consequently the extent of the vasodilatation is less.

Tension Headache

This type of headache is associated with nervous tension or a state of physiologic hypertonicity, which is followed by hypotonicity and subsequent vasodilation, in which state the headache occurs. The headache may be localized or generalized and may involve any one or more vascular areas. A certain degree of nervous tension is frequently associated with the migraine syndrome.

Miscellaneous Unclassified Headaches

This constitutes a very large group of patients in whom the headache may vary from the severe to the mildest type. They may simulate any of the abovementioned types but lack distinct features by means of which they may be classified. As will be pointed out later, they present features of vascular involvement and respond to the same types of therapy.

Wolff[13] and his associates have shown that in migraine the pain is vascular in origin and that there are three phases to consider: (1) The vasoconstrictor phase which causes the scotomas and cortical manifestations. Pain does not occur in this phase of vasoconstriction. A vasodilatory agent may abort an attack at this time. (2) The vasodilator phase. Vasodilation is the immediate cause of the pain, in that pain-sensitive structures surrounding certain vessels are stretched and pulled. Vasoconstrictor agents, such as ergotamine tartrate (gynergen) or dihydroergotamine methanesulfonate (DHE-45), if administered early by injection, will abort the attack in the majority of instances. (3) The edema phase will follow if the vasodilation lasts any considerable length of time and is comparatively resistant to vasoconstrictor therapy.

In migraine the initial vasoconstrictor phase ends in vascular fatigue, producing the state of vasodilation, which becomes increasingly difficult to relieve by vasoconstrictor agents once it has completely developed. This is also true in the tension headache. Practically all the other types of headache apparently do not have a definitely perceptible prevasoconstrictor phase, so the headache begins suddenly with vasodilation. Vasoconstrictor agents are, therefore, the most effective symptomatic treatment for this type.

Considering the fundamental mechanism of the production of headache, it is apparent, as Horton[6] and others have pointed out, that pain occurs in the phase of vasodilation. In migraine and tension headache there is a definite vasoconstrictor phase which precedes the vasodilation. In other types of headache, the vasoconstrictor phase is usually absent or at least insignificant. The slow onset of attacks not infrequently noted in atypical histaminic cephalalgia and in the miscellaneous group suggests, however, that some degree of preliminary vasoconstriction may occur. Much better results with the use of the drug are obtained in those cases in which the preliminary vasoconstrictor phase does not manifest itself. Prolonged vasoconstriction apparently results in vascular fatigue, and when vasodilation develops, it is sometimes difficult to reproduce the degree of vasoconstriction which is necessary to relieve the attack.

In the cases of typical histaminic cephalalgia, and to a lesser degree in the atypical group, the onset of the attack with vasodilation is sudden, unpreceded apparently by vasoconstriction and with

absence of vascular fatigue: hence the prompt and satisfactory relief obtained with cafergot in a very high percentage of cases.

Upon consideration of all these factors, the results may be more accurately evaluated by dividing the cases into three groups as follows: (1) the migraine-tension, (2) the histaminic cephalalgias, and (3) the miscellaneous. The poorest results in our series[5] were obtained in the migraine and tension cases. In group two, excellent results were noted in 90 per cent of the cases of typical histaminic cephalalgia. In the atypical cases, the results were highly satisfactory, but to a lesser degree than in the afore-mentioned group. In the miscellaneous group, which includes unclassified cases, excellent results were noted in 85 per cent.

MIGRAINE AND ALLERGY

A great deal of difference of opinion exists among different observers as to the relationship of allergy to migraine. Some allergists feel that allergy plays the most important part, yet other observers do not even mention allergy in discussing the subject of migraine. In our experience, as pointed out elsewhere, migraine and allergic headache are two unrelated and distinct phenomena. The incidence of allergy among migraine patients is about the same as that among normals.

Some recent comprehensive studies are in agreement with our observations. On the basis of very carefully and well-conducted studies of 40 migraine patients, Hackzell, Kraepelien and Vahlquist[3] concluded that allergy is not a common cause of migraine.

An extensive investigation of the relation of allergy to migraine was conducted by Schwartz.[10] In connection with the investigation of the relation of heredity to asthma, he personally interviewed relatives of 191 asthmatics, 50 patients with baker's asthma and 200 normal persons without allergic disease with regard to the incidence of migraine. A total of 3,815 relatives from four generations were questioned. The incidence of migraine was identical in the two groups, therefore, it has no hereditary relation to asthma. Among 171 migraine patients, no relation of allergy to attacks of headache could be ascertained. Schwartz concludes that it is erroneous to identify a disposition to migraine with an allergic disposition.

ALLERGIC HEADACHE

Headache of allergic origin should be definitely classified as such and not as allergic migraine. The vascular types of headache described above are not allergic, but they may occur in allergic individuals. The following outline[4] of the characteristics of allergic headache may be followed to distinguish it from other types:

1. Family history of allergy usually positive as with other manifestations.
2. Past history of allergy, other manifestations may precede onset of allergic headache.
3. Association of other manifestations of allergy such as urticaria, angioedema, eczema, gastrointestinal allergy, hay fever, and inhalant allergy of the respiratory type.
4. Headache may be associated with cerebral allergy—personality changes, mental confusion, disorientation, etc.
5. Prodromal symptoms usually gastrointestinal upset, vomiting, distention, polydipsia, edema of feet and hands, swelling of face, marked gain in weight, possibly urticaria, first 12 to 24 hours. Visual prodromata not common.
6. Headache usually generalized with sense of pressure, dizziness and mental confusion—cerebral edema.
7. Attack of headache of severe pressure throbbing type not relieved by vasoconstrictors, such as epinephrine or ergotamine, on account of cerebral edema rather than vascular dilatation as in migraine.
8. Attack of headache lasts about 12 to 24 hours. Complete cycle, onset to termination, 48 hours.

9. Terminal phase of attack characterized by polyuria, loss of weight (edema), return to normal.
10. Attack may be prevented by ingestion of adequate dose of antihistamines preceding anticipated exposure to allergen or the preingestion of a protective dose of allergen in the form of diluted extract or as propeptan.
11. Entire syndrome may be reproduced repeatedly with ingestion of causative food.

HISTAMINIC CEPHALALGIA

This type of vascular headache follows a distinct pattern although there are many instances in which many of its features are absent in the commoner atypical types. The following diagnostic points should be noted in history taking.

I. Characteristics.

1. Headache
   (a) Unilateral
   (b) Short duration, generally lasts less than hour
   (c) Begins, and often ends, suddenly
   (d) Awakes patient one to two hours after going to sleep
   (e) May be eased by sitting up, standing erect, or going out into cold atmosphere. Stooping over may increase attack

2. Signs
   (a) Profuse watering and congestion of the eye
   (b) Nasal discharge or stuffiness of the nose
   (c) Sweating on side of forehead or face

   (a), (b), (c): All on involved side

   (d) Increased surface temperature
   (e) Swelling of temporal vessels of side of head

3. Pain
   (a) Very severe, constant, burning, boring
   (b) In the eye, side of face, temple, back of ear
   (c) Attacks very frequent and of long duration, i.e., months or years
   (d) Tenderness over branches of external and common carotid
   (e) Compression of common carotid artery (and occasionally the temporal artery) may give prompt relief

4. Miscellaneous
   (a) Whole head or only occipital region may be involved
   (b) Both eyes may tear
   (c) Right and left sides of head about equally affected
   (d) No G.I. or visual symptoms
   (e) Most have no periodic headaches in earlier years: some may have had "migraine"
   (f) No relation to menstrual periods
   (g) Most patients in fourth, fifth decades of life
   (h) Operative procedures give no relief
   (i) Alcohol may cause an attack, but abstaining does not give a cure
   (j) Spontaneous remissions may occur. During these, alcohol does not produce a headache
   (k) Night characteristics, pain may occur only at night and with clock-like regularity (sometimes only during the day)

VARIABILITY OF HEADACHE PATTERNS

Although certain types of headache, such as true migraine, histaminic cephalalgia and those of allergic origin, may follow more or less definite patterns, a great variety of types which cannot be classified are commonly encountered. They respond, however, to the same therapeutic procedures, and those patients who represent these types should not be denied the benefit which may be obtained from adequate therapy.

In general, it is evident that there is an interrelationship among the phenomena of vasoconstriction, vasodilation, spasm and edema, and that these relationships to a great extent determine the various patterns of headache and related phenomena. In some instances, for example, in histaminic cephalalgia, sympathetic-parasympathetic phenomena, characterized by sweating, increased temperature, lacrimation, nasal congestion and increased secretion may also be present. In those instances in which the constrictor and edema factors are present the therapeutic problem is more difficult to manage. Such cases are repre-

TABLE I

HISTAMINE THERAPY

INTRAVENOUS METHOD

Histamine acid phosphate 2.75 mg.	Controlled administration
1 cc. 1:1,000 = 1 mg. histamine base	Mild flush—no localization of dilatation
IV in 250 cc. saline — 90 minutes	No headache induced

SUBCUTANEOUS METHOD

Dilutions

no. 3 1:1,000 (1 mg. Histamine per cc.)
no. 4 1:10,000 → no. 3 1:1,000 Twice daily injections—Build-up therapy

OPTIMUM DOSAGE METHOD

no. 5 1:100,000	Occasional	
no. 6 1:1,000,000		Precipitation of attack of headache
no. 7 1:10,000,000 ..... Average dose, therapeutic		
no. 8 1:100,000,000		
no. 9 1:1,000,000,000	Rare	

Begin dosage with no. 7, 0.10 cc. 1:10,000,000 (subcutaneous)
Find optimum effective dose
(1) Dose too small—no noted effect
(2) Optimum dose—relief
(3) Dose too large—precipitation of attack of headache
Dosage intervals 3 to 4 days → 14 days → discontinue

sented by the migraine and tension groups. Attacks are likely to be more prolonged than with the vasodilator types.

TREATMENT OF VASCULAR HEADACHES AND RELATED PHENOMENA

The treatment of these conditions is directed to the restoration of normal vascular balance and to the symptomatic relief of the attacks. Before the introduction of histamine and the ergot preparations it was believed that the cause of the trouble was sinus disease or the actual involvement of certain nerve structures, especially the nasal ganglion and the vidian nerve. The relief of an attack of headache usually could be accomplished by cocainization of the nasal ganglion. Varying periods of relief were also obtained following the injection of alcohol into the nasal ganglion. Operative procedures upon the posterior ethmoids and the sphenoids were often followed by dramatic relief, but a recurrence of symptoms usually occurred after some weeks or months. Surgical interference apparently created a local hyperemia and increase in the vascular tone, thus breaking the cycle of attacks. When attacks recurred, it was customary to reoperate, especially if the x-ray showed that a lonely ethmoid cell remained.

TABLE II

USE OF ERGOT IN TREATMENT OF MIGRAINE

METHOD OF ADMINISTRATION	MEDICATION	DOSAGE * AVERAGE DOSE	EQUIVALENT AMT. OF ERGOT
Parenteral	A. Gynergen (Ergotamine tartrate)	*0.5 to 1.0 cc.	0.25 to 0.5 mg.
	B. DHE-45 (Dihydroergotamine)	0.5 to *1.0 cc.	0.5 to 1.0 mg.
Oral	A. Gynergen (Ergotamine tartrate)	5 mg. start 2 mg. every hr. to relief or total of *11	5 to 11 mg.
	B. Cafergot—EC 110 (Ergotamine tartrate 1.0 mg. Caffeine 100 mg.)	2 tablets to start 1 tablet every half hr. to relief or to total of *6	2 to 6 mg.
	C. Ergotamine tartrate 2 mg. Dexedrine 3 mg.	1 capsule every hr. No more than 3	2 to 6 mg.
	D. Bellergal (Bellafoline 0.1 mg. Ergotamine tartrate 0.3 mg. Phenobarbital 20.0 mg.)	1 t.i.d and at bedtime	0.3 to 0.75 mg.
Rectal	A. EC 112 (Ergotamine tartrate 2.0 mg. Caffeine 100 mg.)	*1 to 2 suppositories	2 to 4 mg.
	B. ECB 115 (Ergotamine tartrate 2.0 mg. Caffeine 100 mg. Bellafoline 0.25 mg.)	*1 to 2 suppositories	2 to 4 mg.
	C. ECBP 163 (Pentobarbital sodium 60 mg. Ergotamine tartrate 2 mg. Bellafoline substance 0.25 mg. Caffeine 100 mg.)	*1 to 2 suppositories	2 to 4 mg.
	D. EC 222 (Ergotamine tartrate 2.0 mg. Caffeine 200 mg.)	Under investigation	Under investigation
	E. DHEC no. 1 (Dihydroergotamine methanesulfonate 4.0 mg. Caffeine 100 mg.)	Under investigation	Under investigation

HISTAMINE THERAPY

The final answer to the solution of the problem of vascular headache and related phenomena as we know it today came with the introduction of histamine therapy by Horton and his co-workers[7] in 1939. Other vasodilator agents, especially nicotinic acid, have also been effective in some cases. By some mechanism still unknown, vascular balance may be restored sometimes after a comparatively short course of treatment. Longer or shorter periods of relief may occur without further treatment. A recurrence of attacks, however, is the usual rule.

The various methods of administration of histamine therapy are outlined in Table I. The small optimum dosage type of therapy in our experience has been very satisfactory and best adapted to office practice. In some instances, more satisfactory results have been obtained by adding nicotinic acid in 25 to 50 mg. doses three times daily. We have also employed a combination of 2 mg. of Dexedrine and 25 mg. of nicotinic acid with some success. In some cases in which histamine failed to give results, satisfactory relief was obtained with nicotinic acid.

SYMPTOMATIC TREATMENT

In the symptomatic treatment ergotamine tartrate and its derivatives have been the most effective known agents. Small doses of epinephrine (0.05 to 0.10 cc.) may be effective in aborting an attack. Antipyretics and sedatives are of little value. For the severe acute attack the intramuscular injection of 0.5 to 1 cc. of DHE-45 is very effective, but must be administered early. More recently, various types of suppositories have been introduced for use by the patient when indicated. For the attack of average severity cafergot is usually satisfactory. For the tension cases or more prolonged attacks bellergal is the drug of choice. Table II presents a list of the available products for symptomatic relief.

CASE REPORTS

The following case reports are presented to illustrate some of the patterns of headache and related phenomena encountered in the daily practice of otolaryngology. We wish to emphasize, especially, the importance of recognizing those vascular phenomena which are not confined to the calvarium but are directed to specific areas of the face, the parotid region, the ear, the postauricular area, the posterior and lateral cervical regions, the tongue, pharynx and larynx. These cases used to represent real diagnostic problems, for no pathologic condition of the tissues could be found and nothing was known to relieve the patients' symptoms. We have accumulated sufficient clinical evidence to show that these cases represent vascular phenomena and that satisfactory relief may be obtained with histamine therapy.

Those of you who were practicing otolaryngology previous to 1939 (the prehistamine days) can no doubt recall many of the cases referred to previously in this paper in which no definite diagnosis could be made and in which, in many instances, unnecessary surgical procedures were instituted without relief. At the Mayo Clinic, it was customary to refer most of the patients of this type to the Department of Laryngology, Oral and Plastic Surgery for examination, consequently the incidence of these cases was very high. We were impressed with the large number of patients who had been subjected to some type of surgical interference.

In Greenfield Sluder's clinic a great many headache patients were subjected to the ethmo-sphenoid operation or the injection of the nasal ganglion with alcohol. Sluder's textbook,[11] *Nasal Neurology, Headaches and Eye Disorders,* published in 1927 represents a lifetime contribution to this subject. An amazing variety of case reports are presented. Sluder was a keen observer, an inveterate, sincere worker; he was highly respected and his contributions were internationally known. Unfortunately he did not live long enough to learn the true answer to most of these problems, which came with the introduction of histamine for the treatment of vascular headaches by Horton and his associates in 1939.

A statistical study of our series of cases (more than 600) will be presented

FIG. 1—The carotid and subclavian arteries and their branches (from Cunningham).

1. Superficial temporal artery
2. Internal maxillary artery
3. Posterior auricular artery
4. Occipital artery
5. Internal carotid artery
6. External carotid artery
7. Transverse cervical artery
8. Subclavian artery
9. Transverse facial artery
10. Superior labial artery
11. Inferior labial arteries
12. Submental artery
13. External maxillary artery
14. Lingual artery
15. Superior thyreoid artery
16. Common carotid artery
17. Inferior thyreoid artery
18. Subclavian artery

in a subsequent presentation. The following case reports represent some of the interesting problems encountered.

Anatomical Considerations

The most important arterial branches which may be involved in various vascular pain syndromes are illustrated in figures 1 to 5. Some may follow classical patterns; others are difficult to classify other than to note their relationship to certain arteries. We used to think of pain in relation to the sensory nerves, now we must consider the arteries as well. By careful interrogation as to the exact location and course of the pain, the vessel which is involved should be easily identified. It is amazing to note how perfectly they may correspond.

From a large series of cases we have selected a few which will illustrate some of the points which have been brought out in the foregoing discussion.

Case 1. R.C., a male, 33 years old, reported for observation on February 6, 1941, on account of pain referred to the left upper jaw, the eye and the frontal region. All the characteristics of an acute sinusitis were suggested. Four days previously a left upper second molar was removed. Nothing definite was found to account for the pain, no sinusitis could be demonstrated.

The patient returned for observation three weeks later, but in the meantime Horton's article[6] on histaminic cephalalgia had been read. Further interrogation of the patient revealed a typical histaminic cephalalgia on the left side of five years' duration. The patient presented all the classical symptoms, such as the deep boring pain in the eye, frontal region,

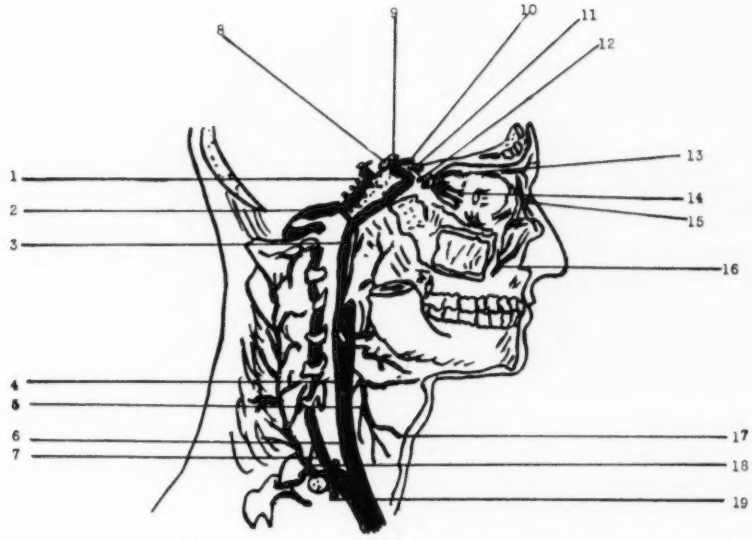

FIG. 2.—The carotid, subclavian and vertebral arteries and their main branches (from Cunningham).

1. Basilar artery
2. Vertebral arteries
3. Internal carotid artery
4. External carotid artery
5. Superior thyreoid artery
6. Common carotid artery
7. Vertebral artery
8. Stalk of hypophysis
9. Middle cerebral artery
10. Anterior cerebral artery
11. Optic nerve
12. Ophthalmic artery
13. Ethmoidal arteries
14. Ciliary arteries
15. Nasal artery
16. External maxillary artery
17. Crico-thyreoid artery
18. Deep cervical artery
19. Thyreo-cervical trunk

cheek and upper teeth, with lacrimation of the left eye, and sweating of the left brow (fig. 1). The onset and cessation of attacks were sudden. They lasted 30 to 40 minutes and attacks occurred at night after retiring as well as during the daytime. Treatment with 1:10,000,000 or no. 7 histamine produced immediate relief, later complete remission for indefinite periods.

For us this was the beginning of the study and observation of a large group of vascular phenomena which represents some of the most interesting problems in otolaryngology, the solution of which has produced an incalculable degree of personal satisfaction to us as well as to the patient.

In typical histaminic cephalalgia, the external carotid is involved. Since the pain in this type is localized deep in the eye and the upper teeth the internal maxillary branch of the vessel is also involved (figs. 3 and 4).

Case 2. J.M.H., a male, aged 47, reported for examination on January 18, 1944. On this day he was on his way to a hospital to have an intracranial section of the right trigeminal nerve for relief of attacks of severe pain referred to the region of the right eye, frontal and cheek area. Occasional attacks occurred on the left side. For many years the attacks would occur for only a period of four to six weeks out of the year. Before coming under our observation, repeated attacks occurring night and day had been present for six months, but previous to that time he had had no attacks for six years.

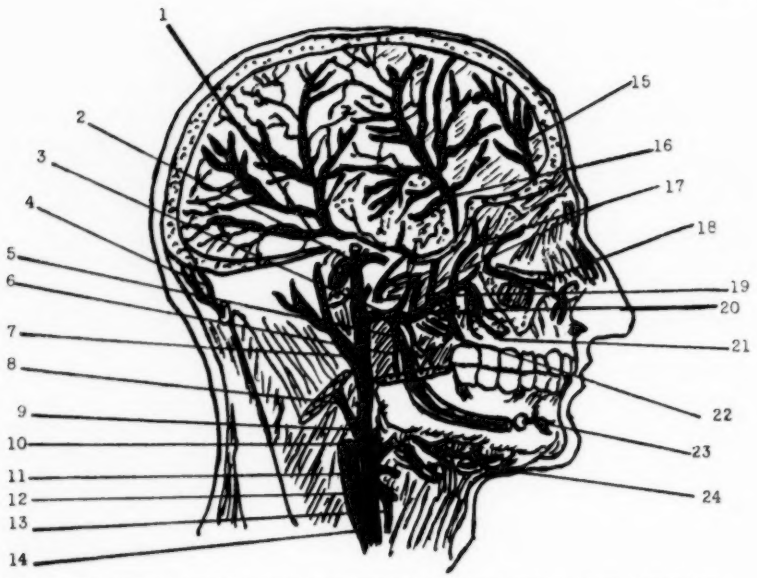

FIG. 3—The external carotid, internal maxillary and meningeal arteries (from Cunningham).

1. Posterior branch of middle meningeal artery
2. Superficial temporal artery
3. Internal maxillary artery
4. Occipital artery
5. Inferior alveolar artery
6. Mylo-hyoid artery
7. Posterior auricular artery
8. Occipital artery
9. External carotid artery
10. External maxillary artery
11. Lingual artery
12. Internal carotid artery
13. Superior thyreoid artery
14. Common carotid artery
15. Anterior meningeal artery
16. Anterior branch of middle meningeal artery
17. Deep temporal arteries
18. Infraorbital artery
19. Middle meningeal artery
20. Accessory meningeal artery
21. Posterior superior alveolar artery
22. Buccinator artery
23. Mental artery
24. Submental artery

The history brought all the characteristics of a typical histaminic cephalalgia, such as lacrimation, sweating, night attacks, sudden onset and cessation, severe, deep, boring pain in the right eye, frontal and cheek areas. There were no trigger areas, paroxysmal pains or other features of trigeminal neuralgia.

We have encountered four patients who had section of the trigeminal nerve for this condition with no relief. The routine treatment with histamine no. 7 and no. 6 gave complete relief (Table I).

Treatment may be discontinued in these patients as soon as a period of complete relief of two weeks is obtained. Long remissions free of symptoms, sometimes for years, are characteristic of the course of this type of vascular syndrome. A course of treatment "breaks the cycle." Any operation which increases the blood supply in the affected area will do the same, hence the misleading good results sometimes obtained from surgical interference.

Case 3. J.S., a male, aged 45, was first observed in May 1938 for attacks of severe pain in the left eye, frontal and cheek areas. All the other features of histaminic cephalalgia were present. This patient had the very best medical service available at that time. Section of the trigeminal nerve on the left side

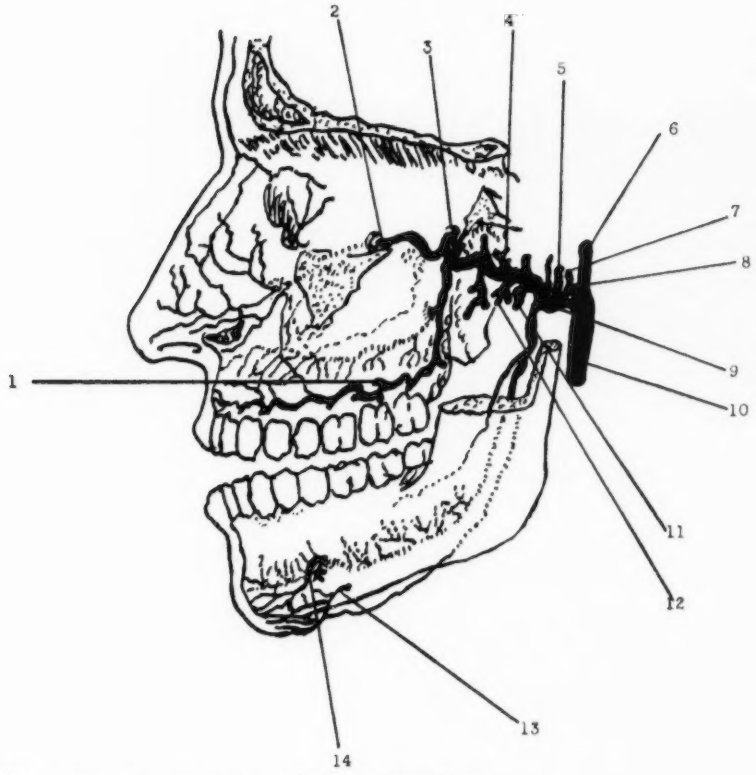

FIG. 4—Internal maxillary artery and branches (from Deaver).

1. Gingival branch of posterior dental artery
2. Infraorbital artery
3. Sphenopalatine artery
4. External pterygoid artery
5. Middle meningeal artery
6. Superficial temporal artery
7. Tympanic artery
8. Deep auricular artery
9. Internal maxillary artery
10. External carotid artery
11. Masseteric artery
12. Inferior dental (inferior alveolar) artery
13. Submental artery
14. Mental artery

was considered, but instead a few other operations were performed: avulsion of the left supraorbital nerve, removal of the tonsils, extraction of teeth and one nasal operation. Finally an intracranial procedure was undertaken on the trigeminal nerve and regional sympathetics. As a result of this procedure, the patient lost the left eye. This "broke the cycle" of his pain attacks.

We saw this patient in 1935, which preceded our understanding of histaminic cephalalgia and its treatment by six years. Then contact with the patient revealed that all the above surgery had been performed.

Case 4. Mrs. L. J., 51 years old, was seen for the first time on November 21, 1946, with a typical history of histaminic cephalalgia on the right side of twelve years' duration. In spite of the absence of any diagnostic evidence of trigeminal neuralgia, a neurosurgeon performed an intracranial section of the right trigeminal nerve in 1937. She had no symptoms for two years, since the operation "broke the cycle of attack," but after that the typical attacks reappeared.

Complete relief of symptoms occurred after a short course of histamine therapy (no. 6 dilution). Relief lasted six months. Then a few

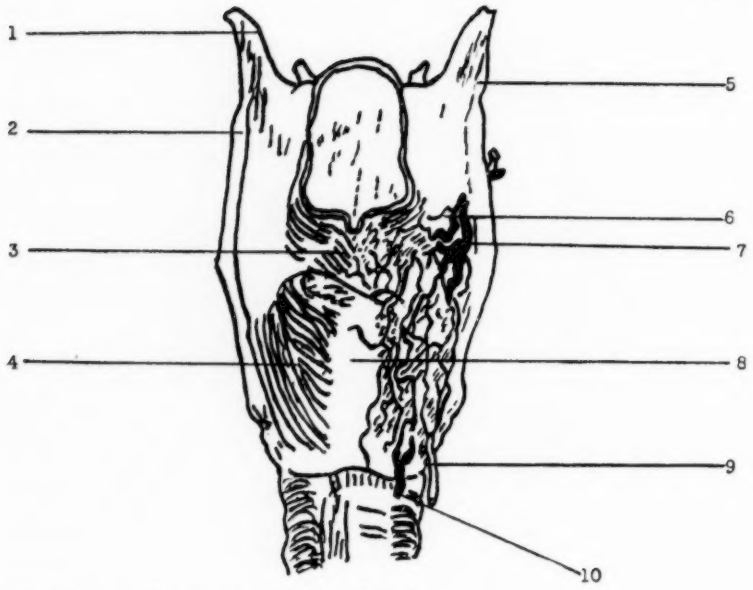

FIG. 5—Nerves and arteries of larynx (from Deaver).

1. Greater cornu of hyoid bone
2. Superior cornu of thyroid cartilage
3. Arytenoideus muscle
4. Posterior crico-arytenoid muscle
5. Cartilago triticea
6. Internal laryngeal nerve
7. Superior laryngeal artery
8. Cricoid cartilage
9. Recurrent laryngeal nerve
10. Inferior laryngeal artery

more treatments were followed by six months' relief again. Until the fall of 1951, only sporadic attacks occurred for a period of four years.

Case 5. C.H.J., a male, 47 years old. On January 12, 1952, the following letter was received,

January 10, 1952

Dear Doctor Hansel:

My physician, a nose and throat specialist, has suggested that I get in touch with you in connection with some sinus difficulty I have experienced over the last several years. He thought that perhaps you would feel that a trip to St. Louis to consult with you might prove desirable.

To begin chronologically, for a good many years (nearly 20) I have been troubled with a tendency to be sleepy during the day, especially when trying to read. My eyes have felt tired, and there has been a great desire to close them. Sometimes my eyes have been somewhat inflamed, with a persistent dull pain behind the right eye which made it difficult to concentrate. Along with this has been a constant postnasal drainage.

I have consulted a great number of reputable internal medicine doctors, eye and nose and throat specialists, and the following measures taken over the last 20 years at the suggestion of these men to overcome the trouble:

1. Removal of infected tonsils, 1928.
2. Removal of infected appendix, 1937.
3. Nasal submucous resection, 1941.
4. Removal of cyst in right maxillary sinus, 1943.
5. Opening and draining of right sphenoid sinus, 1947.
6. Large window for ventilation and drainage made in anterior wall of right sphenoid sinus.

Item 5 above gave complete, but only temporary relief. Item 6 also gave complete relief, with entire disappearance of the ocular symptoms, but again this was only temporary, as the opening made in the operation closed up before the infection was entirely cleared up

and today I am as badly off as ever. Since the doctor has died in the meantime, I have been unable to return to him for further treatment. Several separate times I was subjected to extensive clinical examinations — blood work, basal metabolic rate, gastrointestinal tests, x-rays of skull and chest, eye examinations and refractions, etc., none of which disclosed anything of value in solving my problem.

Several attempts have been made to discover if my symptoms were really allergic in nature. Skin sensitivity tests some years ago indicated that I was sensitive to tobacco (which I do not use), but that was about the only item that showed response. The conclusion was that it was not a food allergy. Nevertheless, I eliminated certain items from my diet, eggs, milk and dairy products, chocolate, etc., but could see no improvement.

Another approach through antihistaminics was tried, using Pyribenzamine ephedrine, chlortrimeton, Phenergan by mouth, and other antihistaminic eyedrops, and Cortisone, all with no observable effect. Another treatment tried about a year ago made use of hypodermic "shots" over a period of several months, also with no effect.

I am getting very discouraged. My job is being affected and I am getting to be an irritable person to live with. Just recently I had a terrific infection again in my right sphenoid sinus. My attending physician feels that there is little more that he can do, but suggested that I write you, which brings us back to the start of this long letter.

(1) Do you feel that it would be worth while to pursue the allergy angle further in my case? I have tried to present reasonably briefly the facts in this long medical history so that you may have some basis for judgment.

(2) If you do feel this is desirable, and I came to St. Louis to consult you approximately how long would such a process take, and about how expensive would it be? I have a rather ordinary position here in a metallurgical capacity, and would have to make arrangements to be absent for the required time. Would hospitalization be involved?

My physician feels that if my case is really allergic in nature and not just one of simple infection, you are the one best fitted to diagnose it. In the event that a series of treatment over a considerable time period is prescribed, he would be most willing to cooperate by administering them under your direction.

May I hear from you soon on this matter, which is most important to me? Thanks.

Very truly yours,
C.H.J.

This case is presented to illustrate again the variety of surgical procedures to which these patients with vascular headaches are subjected. This patient presented no history or any evidence of having had any definite disease process in the sinuses. Although he did not present all the signs of a typical histaminic cephalalgia, his symptoms were vascular in nature. The relief from the operations on the sphenoid is typical of a nonspecific effect on the vascular mechanism.

Very prompt relief of the pain and sleepiness was obtained with histamine therapy (no. 7 dilution).

Case 6. Mrs. H.R., aged 50, was observed for the first time on March 1, 1951, complaining of sinus trouble, headaches and arthritis of eight years' duration. She had had three attacks of facial paralysis (Bell's palsy); two on the right, one on the left. For five years she had had typical histaminic cephalalgia on the left side with pains in the frontal, temporal and deep ocular regions. She also had lacrimation, stuffy nose and sweating on the left side. More recently the attack of pain began with a tenderness and swelling of the left side of the neck. The pain radiated upward to the temporal and mastoid areas. Observation during an attack revealed a visible swelling of the left common carotid area. The vessel was tender to touch and pressure. This was our first observation of this phenomenon. The pain distribution may be traced in figure 1.

Response to histamine therapy was very prompt. Incidentally her rheumatoid arthritis promptly cleared and has remained so for six months with no further treatment following a few injections of 1/10 unit of staphylococcus toxoid.

Case 7. N.W., male, aged 45, reported for examination of February 20, 1952, complaining of attacks of pain of eight months' duration referred to the deep zygomatic and temporal area on the right side. The attacks began and ended suddenly. The pain also extended into the upper jaw and teeth. Satisfactory relief was obtained with histamine therapy (no. 7).

The distribution of pain in this case appears to follow the internal maxillary artery as illustrated in figure 4.

Case 8. Miss E.E., aged 34, was first observed on September 16, 1950, giving a history of pain in both sides of the face of five years' duration referred to the region of the canine fossae. Pain was aching in type, coming on in attack and lasting for several hours. Nothing was found to account for the pain. Satisfactory results followed histamine therapy.

The arterial involvement in this case was probably localized in the terminal branches of the infraorbital artery in the canine fossae, as illustrated in figure 4.

Case 9. Miss K.N., aged 58, reported for observation on August 10, 1950, complaining of attack of burning and a sense of constriction in vertex area on each side. The attacks occurred three or four days a week, off and on, for a period of eleven years. Histamine and nicotinic acid therapy produced almost complete relief.

The arterial localization in this case appeared to be either in the terminal branches of the temporal artery as shown in figure 1 or in the branches of the meningeal arteries as illustrated in figure 3.

Case 10. W.P., a male, aged 61, reported for observation July 14, 1949, giving a history of typical histaminic cephalalgia on the right side of one year's duration. The attacks of headache occurred several times a week. About once every two weeks, attacks of sudden complete blindness lasting ten to fifteen minutes occurred in the eye on the same side, but never with the attacks of headache. Both the attacks of headache and the blindness subsided with histamine therapy.

The attacks of blindness were caused by involvement of the ophthalmic artery (central artery of the retina), a branch of the internal carotid as shown in figure 2.

Case 11. Mrs. A.H., aged 34, reported for observation on September 13, 1946 presenting all the typical characteristics of histaminic cephalalgia of about twelve years' duration, associated with a sense of fullness of the ear on the same or right side. She also had attacks of pain in the right arm and at times in the calf of the right leg. During two pregnancies she was completely free of symptoms. With histamine therapy the headaches, the fullness in the ear and the pains in the extremities subsided.

The ear symptoms in this case were probably caused by involvement of the arteries supplying the eustachian tube, branches of the internal maxillary artery. The pain which is referred to as deep in the eye also must come from this vessel (fig. 4).

Case 12, a female, aged 40 years. This patient was seen in consultation with Dr. B. Barrett in Pensacola, Florida, on January 22, 1951. She complained of headaches and vertigo, which responded promptly to histamine therapy. The patient volunteered the statement that with the relief of the above symptoms she no longer had attacks of pain or cramps in the calf of the leg which came on at night during sleep. Interrogation of the patient suggested that the type of pain, the sudden onset and cessation, the relief following exercise of the leg were characteristic of a vascular type of phenomenon.

These cases indicate that pain of vascular origin may occur in other parts of the body associated or unassociated with headache. Osler's abdominal migraine is of vascular origin. It is not unreasonable to assume that in certain cases precordial pain, back pain, and pelvic pain may be of vascular origin. These possibilities appear to be worthy of consideration. Their association with headache should be recognized and the effect of histamine therapy should be noted.

The observation of patients with vague pains in the tongue, the pharynx, and larynx is not an uncommon occurrence in daily office practice. In the past, these patients always presented a diagnostic puzzle as no pathologic condition could be found to account for the complaints, no effective treatment was

known, consequently they were relegated to the classification of "neurotics." The following case reports present convincing evidence that these phenomena are vascular in nature.

Case 13. J.J., a male, aged 56, was observed for the first time on February 12, 1946. He complained of a sense of tightness and discomfort in the left laryngeal region of three months' duration. At times there was also a sense of soreness in the tongue. The attacks would come on at night after going to sleep. Activity gave relief. Treatment with histamine (no. 6) gave complete relief for nine months when he returned with soreness referred to the tongue. Again treatment was effective for five months, then four months. For five years since then, there has been no return of symptoms.

The vascular involvement in this case appeared to be in the superior laryngeal artery (fig. 5) and in the lingual artery (fig. 1).

Case 14. Mrs. G.K., aged 58 years, reported for observation on September 5, 1951, complaining of a tender or sore area in the left side of the upper laryngeal region of many years' duration. The symptoms would recur without cause and were better when she was active. She also had attacks of pain referred to the left cheek area. Treatment with histamine produced almost immediate relief, and after four weeks there were no more symptoms for a period of five months.

In this case, the symptoms were referred to the left superior laryngeal artery, as in the preceding case. Mild involvement also occurred in the left upper maxillary area (fig. 4).

Case 15. Mrs. M.B., aged 70 years, reported for observation on September 28, 1951. She stated that for about twenty-five years she had suffered with attacks of "biting pains" first on one, then the other, side of the neck, but mostly on the left, referred to the upper laryngeal region. Attacks would sometimes occur at night and awaken her from sleep. She was better when out of bed. She had never been free of attacks for longer than one month in twenty-five years. Within two weeks the symptoms were entirely relieved with histamine therapy and there was no return of attacks after three months.

Although this case is similar to the two preceding cases, the patient had almost continuous discomfort for twenty-five years. She had many examinations and treatments with no relief.

Case 16. W.L.M., male, aged 52, was observed on January 10, 1947, complaining of attacks of pain in the deep muscles of the back of the neck, mostly on the left side, of one year's duration. The deep boring pain would sometimes occur at night. Treatment with histamine (no. 6) by injection and by sublingual administration (no. 4) gave prompt relief, and after four months there was no recurrence of attacks.

In this case the deep cervical artery was no doubt the vessel involved (fig. 2). In many cases of so-called cervical myositis the underlying process is probably of vascular rather than of muscular origin.

DISCUSSION

These atypical cases have been presented to illustrate the great variety of vascular phenomena which may be encountered in the head and neck. In taking the history of the patients' complaints it is important to obtain all details as to the nature and the exact location of the symptoms. An effort should be made to localize the involvement according to the distribution of the arteries affected.

The atypical cases of vascular disorders are more common than the typical. Not more than 15 to 20 per cent of the cases can be classified as typical histaminic cephalalgia or migraine.

A great many cases of vertigo, tinnitus and sometimes deafness may be included in this vascular group and should be given a trial with histamine therapy in the absence of definite pathologic findings. Bell's palsy has also been successfully treated with histamine.

It is significant that migraine is probably the only type of vascular headache that occurs in childhood and early life. All other types of vascular phenomena occur in the older age group, rarely before the age of 25 years and usually after 40. It is not usual, however, for histaminic cephalalgia to be preceded by true migraine, which usually subsides at about the age of 45 to 50.

We are fortunate in having available a variety of ergot compounds and combinations which give satisfactory symptomatic relief of attacks which may occur until adequate histamine therapy has been instituted.

In the primary management of these cases, diagnosis is important. Any pathologic condition which may contribute to the cause of attacks should be considered. Pathologic conditions may exist which have no relation to the patients' complaints. Above all, it is important to avoid the mistake of performing some surgical procedure which does not relieve the patients' symptoms. This point has been well illustrated in some of the case reports presented here.

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VASCULAR HEAD AND NECK PAIN

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LET US marshall the observed clinical facts relative to vascular head pain—a chronic and recurrent problem, commonly misinterpreted as sinusitis. As an example vessel, we must select an artery which can be kept under tactile observation because it is recognized that the bulk of clinical information gathered about the subject of vascular head pain is purely subjective patient description. The anterior temporal branch of the superficial temporal artery is not an impressive vessel in size but it is representative of the average stem from which vascular pain arises. It is superficially located beneath the skin of the temporal region and its peregrinations can be followed objectively and correlated with the subjective expression of the vasodilating phenomenon.

The pain period is preceded by a prodrome of variable duration—the patient knows he is soon to have severe pain by a definite sensory change in the area, This may be referred to as the ischemic period. There is usually little surface expression of it—collateral supply in the region is good. Soon pain along the course of the artery begins and mounts in intensity—oftentimes rapidly. The artery becomes distinctly palpable. As the full throbbing intensity of pain reaches its apex, the pressure of touch along the course of the artery is ill-tolerated. The vessel is noted to be not only enormously dilated and pulsating but, in addition, the character of the vessel wall is changing. It is becoming thickened, edematous. It is becoming—through increase in thickness and, presumably, length—tortuous. It is visible as a tortuous, pulsating snakelike contour. The pain is excruciating, unbearable, ill-relieved by opiates. The patient knows gentle, external supportive pressure is as kindly treatment as any. There may be nausea and other reflex neuromotor reaction, such as sweating, flushing, and pallor.

As the artery subsides, the pain subsides. For long hours afterward, however, the doughy edematous vessel wall remains palpable and exquisitely sensitive to slightest touch. The brushing of the overlying hair, for example, wakes a miserable soreness.

Whence emanates this disturbed vascular physiology? It comes from irregularities in immunologic, autonomic and endocrine mechanisms which control it. Vascular balance is a state of homeostatic bliss for the individual's vascular and extravascular fluids. When this balance is lost, the individual becomes a transudate problem. When the imbalance relates to the vasa vasoral supply of the branches of the carotid tree and to the arteriolar periphery of those branches, the individual becomes a vascular head and neck pain problem.

The basal nuclear aggregates of the brain stem are of prime importance in maintaining vascular balance. Inherently some are of poor stuff and, hence, the importance of inheritance and family history of vascular imbalance in clinical investigation. Also, equally effective neural and endocrine mechanisms subjected to very different degrees of environmental stress break down to a state

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of imbalance and peripheral expression in proportion to the load they bear. What comprises environmental stress? Principally it is the physical and emotional climate in which we live. However, inhalant and ingestant entities are truly environmental also.

In the area of our disturbed subject artery, what vascular disturbance produces edema and dilatation of its wall? In part, the answer, incongruously enough, is vasospasm—arteriolar spasm. The functional level of the vascular bed is the arteriolar-capillary-venular unit. The vascular anatomy we teach students is purely a system of highways meaningless without these vascular units in the periphery. It were better if we taught them the function of these nameless units and forewent the pleasure of lurid freshman ditties to systematize the main roads in their minds.

When an arteriole goes into spasm whether the stimulus be from central impulsion or local axon reflex or antigen-antibody interplay at the smooth muscle sphincter an interesting sequence of events is initiated. The dependent capillary dilates, possibly due to ischemic damage to the endothelium and the presence of uncontested reflux venous tonus, and fluid transudate leaks between separated endothelial cells into the tissue spaces. This then is the motor unit of wheal formation, the dynamic origin of edema. The biomicroscope has demonstrated it sufficiently frequently so doubt as to its correctness no longer exists.

In addition, the primary status of the capillary endothelium, the electrolyte balance between vascular and extravascular spaces, and lesser known variations in tissue cells themselves have an exceedingly important place in maintaining fluid balance. These factors are, to a great degree, dependent upon endocrine balance. Imbalance in gonadal, thyroidal or adrenocortical hormones can establish a circumstance of easy transudate which alone or in combination with the neurovascular or antigen-antibody inspired arteriolar spasm can set in motion the sequences of vascular pain.

Where are the arterioles in subject artery whose activity effects the dilatation of the main trunk? They can be of two kinds. In an artery's peripheral bed simultaneous constriction erects a formidable peripheral dam and calls for a proximal circulatory shunt. In the absence of shunt, pulsating arterial force butting a peripheral dam exerts a dilating pressure against the banks of the main stream. Simultaneous with this development one may expect ischemic aura from the tissues of the periphery if collateral arterial supply is not adequate. Peripheral autonomic innervation provides an anatomic basis for this segmental arteriolar behavior. The commonplace blanched single phalanx is a case in point. The second, and probably more important, arteriolar group involved in the vasodilating phenomenon, however, is that of the vasa vasorum of the subject artery wall. The integrity of vascular intima, media and adventitia is solely dependent upon the behavior and health of the vasa vasoral arteriolar-capillary-venular complexes. The initial contraction of main arterial smooth muscle produced by vasa vasoral spastic ischemia cannot endure for long. The inevitable subsequent arterial muscular flaccidity and edema in the presence of enduring ischemia and easy transudate when impinged by the shunt complexities detailed above lays the mechanical ground work for dilatation of the arterial trunk. The edema of the subject artery wall resulting from this vascular crisis and transudate of the vasa vasoral arteriolar-capillary-venular periphery is that which is felt as a doughy thickening of the arterial wall beneath the temporal skin.

In organizing medical observation and fact into a systematic collection for transmission to others we exhibit a most unfortunate human tendency to pigeonhole. There comes a point at which the average human brain runs out of slots. The folly of expounding a clinical syndrome expressing the pain of every individual branch of the carotid arterial tree must be apparent. This is particularly true in that all combinations of segmental vascular functional misbehavior occur, and in the same individual over many years the pattern tends to be a shifting one. Hence, one ends with more atypical syndromes than typical. The subject must be taught and dealt with as a vascular imbalance with its expression a vasodilating pain and its locale limited only by the anatomic location of the peripheral branches of the carotid artery. The fog of typical and atypical migraines, histaminic cephalalgias, pseudosinus headache, and atypical neuralgias is thus swept aside and the simple mind can go forward to meet disturbed vascular physiology in whatever locale it may be found.

In this same manner it is apparent that many of the clinical phenomena we see and relegate to the "itis" or "idiopathic" categories have their source in the functional behavior of the arteriolar units. Segmental behavior can produce syndromes ranging from tic to epilepsy. It is a fascinating horizon and need not be limited to the pain phenomena of the dilated and edematous arterial wall.

How can intelligent therapy be projected for the edematous arterial wall and distal arteriolar dam if the mechanism in the individual case is not identified? The futilty of a stereotyped therapeutic approach to vascular head and neck pain seems devastatingly clear.

PATHOLOGIC FINDINGS IN ALLERGIC DISEASES OF THE EYE

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THE pathology of allergy of the eye is basically the same as that of any other part of the human body. It deals with the reaction of the cell to a specific irritant or poison. Actually the process is based on the antigen versus antibody reaction within the individual cell. The train of events follows a familiar pattern. The eye becomes irritated and congested, followed by photophobia, lacrimation and the production of mucoid secretion. The conjunctiva thickens into a velvety pale membrane covered with mucoid material which clings to an applicator in strings. Specific bacteria are not present in the smears taken at this early stage but mucus and eosinophils abound. As the condition progresses, invading bacteria may be present but the outstanding changes are in the conjunctiva itself. This tissue becomes hyperplastic with the formation of fairly dense connective tissue rich in eosinophils and plasma cells and covered with a thickened epithelium.

Vernal conjunctivitis or vernal catarrh is the classic allergic disease of the eye and is so named because of its seasonal incidence. The attacks occur in warm weather and become less acute as winter approaches. Treatment is unsatisfactory in many instances, but the condition seems to respond to hormonal therapy, which is a definite improvement over former remedies.

The pathologic findings in vernal catarrh are concerned with the conjunctival smear and the examination of tissue from the catarrhal proliferation.

A conjunctival smear consists of mucoid material spread evenly over a microscopic slide and stained either by Wright's method or with the Giemsa technic. The field examined will show considerable mucus with many eosinophils scattered throughout the area. Usually, no bacteria are found in the early stages of the disease.

Examination of the tissue from the plaquelike proliferation of conjunctiva stained with hematoxylin and eosin shows thickening of the epithelium and papillary hypertrophy in the early phases, followed by fibrosis and signs of chronic inflammation in the later stages. The supporting tissue is rich in eosinophils, contains plasma cells and lymphocytes and shows evidence of fibrous tissue formation. Many of the plasma cells are undergoing necrosis, probably from toxins, which results in the formation of hyalin and produces the milky appearance of the conjunctiva. As the "cobble-stone" plaques become older and the circumcorneal region is affected, the fibrous tissue formation becomes more dense and the secretory epithelium changes its character. In some areas, columnar cells are replaced by flattened elements. The normal secretions of the conjunctiva become altered and the cornea is affected. Sections from these older plaques show more fibrosis, greater epithelial changes and a preponderance of plasma cells and eosinophils.

In addition to vernal conjunctivitis, such conditions as phlyctenular conjunctivitis and the various granulomatous conditions of the eyes are thought to have allergic phases.

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Phlyctenular conjunctivitis is characterized by the formation of one or several pustules at the limbus of the cornea and is aggravated by tuberculoprotein. The condition usually occurs in undernourished, debilitated individuals, mostly children, and responds to improvement in diet and hygienic conditions. Since the disease usually responds to therapeutic measures, there is no excuse for biopsy. However, Leber and Wintersteiner[1] stated that the infiltrating cells are mostly polymorphonuclear leukocytes with some lymphocytes. In the periphery of the ulcers, epithelioid and giant cells are present. Although no caseation is evident, the reaction resembles tuberculosis. This affection is considered to be allergic in spite of the lack of evidence.

The common granulomas of the eyes which show some allergic phases at times are the chalazion, tuberculosis, sarcoidosis, sympathetic ophthalmitis, syphilis and leprosy.

The chalazion is a granuloma of the meibomian gland resulting from acute inflammation followed by attempted resolution. It is characterized by a chronically inflamed cystic wall containing cheesy material, a product of the gland, and degenerated epithelial and inflammatory cells. The reaction of the tissue around the cyst resembles tuberculosis in that there is lymphocytic infiltration with fibrosis and a generous deposition of giant and epithelioid cells. The response of the tissues in the eyelid to the chalazion are said to be allergic despite the fact that eosinophils are difficult to find. It is possible that this is a reaction to retained foreign material which is considered allergic in action by some authorities.

Tuberculosis of the eye often involves the uvea before other structures are invaded. The disease is characterized by discrete or confluent tubercles of the choroid, ciliary body or iris. The tubercles consist of aggregations of lymphocytes and plasma cells surrounding an area of necrosis. Occasional eosinophils are intermingled with the granulation tissue. Epithelioid and giant cells are present in the inflammatory rim of the tubercle. Caseation results from destruction of the nuclei of the cells because of the lack of nutrition. One of the important characteristics of ocular tuberculosis is its tendency to destroy any and all structures, whereas other granulomas respect certain structures. An example is the sparing of the choriocapillaris by the granulation tissue of sympathetic ophthalmitis. Tuberculous tissue will show typical bacilli with acid-fast technic, but other granulomas, with the exception of leprosy, are free of such organisms.

Involvement of the iris and ciliary body with tuberculosis usually spreads by extension into the cornea producing the condition known as deep sclerosing keratitis. The sclera, the most resistant tunic of the eye, is next involved, producing a protuberance of this structure of staphyloma. In some cases the cornea and sclera become necrotic and rupture with the eventual loss of the eye from panophthalmitis.

Sarcoidosis presents essentially the same microscopic findings as tuberculosis without the bacilli and the caseation necrosis. The clinical appearance of the lesions is that of small, discrete nodules in the ocular tissues without signs of intense inflammation or complaints of much pain. Examination of the tissue fails to show necrosis but new fibrous tissue is intermingled with epithelioid and giant cells. Numerous lymphocytes and plasma cells complete the picture.

Sympathetic ophthalmitis is the mysterious inflammation of a sound eye following a perforating injury of its fellow eye. Several theories have been advanced as to the cause of the disease.

The allergic response of the uveal tissue of the good eye to the inflammation of the injured one has occupied an important position in the theories advanced as to etiology. Unfortunately, no definite cause for sympathetic ophthalmia has been discovered and consequently most of the patients become blind unless the injured eye is enucleated as soon as the diagnosis is made.

The microscopic appearance of the granulation tissue of sympathetic ophthalmitis is the same as that of tuberculosis without necrosis. It differs in that it is confined to the uvea and perforations of the sclera and does not destroy adjacent tissues. The granuloma may extend through the perforating wound but will not involve the cornea or sclera.

The distinctions between sympathetic ophthalmia and tuberculosis of the eye is summarized below:

Sympathetic Ophthalmia

History of injury
No caseation as a rule
No destruction of other tissue
Nodules of Dalen on choroid
No histolysis
May attack entire uveal tract
Choriocapillaris not involved
Emissaria involved
Pigment epithelium usually intact
Deep process
Entire iris infiltrated with lymphocytes
Iris tends to bulge forward, resembling iris bombé

Tuberculosis

No history of injury
Caseation usually present
Adjoining tissues destroyed
Tubercular nodules on choroid
Histolysis present
Particular affinity for iris
Choriocapillaris destroyed
No emissarial involvement
Pigment epithelium often destroyed
Superficial and mostly anterior
Iris infiltration more of a nodular type
Sclera may be involved

It is claimed that syphilis of the cornea in the form of interstitial keratitis is an allergic reaction. The microscopic examination of such specimens shows minute blood vessels entering the periphery of the cornea in its deeper layers with chronic inflammatory reaction and a preponderance of lymphocytes and plasma cells. A few eosinophils may be identified. If the cornea reacts unfavorably to antisyphilitic treatment, it may undergo ulceration and necrosis causing the loss of the eye. The granulomatous reaction of the cornea is not as severe as that of tuberculosis or sympathetic ophthalmitis.

Leprosy of the eye may assume tuberculous-like processes in which there is ulceration and necrosis. It may involve only the nerves of the eye for many years, eventually developing the characteristics of a true granuloma. No tissue of the eye is immune and bacilli are recognized with the acid-fast stain. Due to the peculiar degeneration of its cells with fatty impregnation one is often able to identify the so-called foam-cells which are somewhat characteristic of the disease.

The above discussion has been offered to point out some of the characteristics of tissue in conditions which are designated as allergic in nature. It is obvious that antigens and antibodies are not recognized with the ordinary microscope and that swollen and weeping tissues are not excised for histologic examination.

REFERENCE

1. Leber and Wintersteiner: Quoted by Parsons, J. Herbert: The Pathology of the Eye, vol. 1, pt. 1, p. 77, New York & London, Hodder & Stoughton, 1904.

ALLERGY OF THE CORNEA AND BULBAR CONJUNCTIVA

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It is extremely important that we all realize that allergy as we now know it has come of age very rapidly, much more rapidly than many other phases of medicine. Instead of the usual fifty, one hundred or two hundred year acceptance period, the allergic states have been quickly accepted as a strong and possible allergic factor in disease. We must then accept them and do what we can to help the unfortunate sufferers from allergies instead of bickering and discussing, "Why do we have allergy?" or "Is it psychosomatic or is it a chemical process?" When you see a patient whose eyesight has been lost because of neglect from an allergic manifestation, you appreciate the fact that many such people are not being properly cared for.

A patient came to us because of a badly infected right eye. The condition was the result of typical prolonged treatment with a substance to which the man was sensitive. This substance was Butyn. With withdrawal from Butyn we were able to send him back to work. However, his vision had been reduced to 20/200, because of a diffuse corneal scar. Economically, the eye was lost. The allergic reaction may have been to the ointment base or vehicle. The vehicle frequently causes the allergic response. Many drugs can be used in liquid form and not in salve form.

Some time later, this man had another foreign body in his eye, and he was again treated with Butyn, this time in both liquid and salve form, and he was given the two to take home. Actually, this man lost his entire economic vision because his second eye reacted in the same manner as the first one. He will make a good patient on whom to do a corneal transplant, and if one can guarantee him good results with corneal transplants, everyone will be happy. But persons who have this sort of corneal reaction do not always do well with corneal transplants. The new tissue itself may respond like the original and result in a corneal scar.

A new industry has come into our territory that uses beryllium. We have seen several cases of reaction to beryllium fumes or substances used in the beryllium plants, in which the conjunctiva was brick red. Recently an article came out on the use of beryllium and its factors in general medicine. I read the article through very carefully, thinking the authors would make some notation on the effect on eyes, but not a single word was said about our patient, who was included in their list.

As one considers the ocular conjunctiva, almost anything that an individual comes in contact with, various foods or other substances, may produce severe local response. One of our patients had terrific local reaction and circumcorneal injection and infiltration of his cornea. One or two such attacks will not do a bit of harm. The vessels or vascular spaces will return to normal and the cornea will be as clear as ever. But if patients have repeated attacks or repeated insults, there is an actual infiltration of the cornea with vascularization, and it is this type of vascularization that is well treated by beta irradiation or any irradiation. Even contact x-rays in small doses will do it.

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The local use of Cortisone, Antistine or Benadryl and the general use of Pyribenzamine and rest make these people much more comfortable and will frequently clear the eye up until they go into a second attack. The important thing to remember is that Cortisone and the rest of these substances merely stymie the reaction and it is up to you to find out what causes it, not just treat the patient and let him go on his way, because he will have a second and a third attack. Each one might be lessened in severity, but they may go on to corneal vascularity, which will definitely give you trouble.

There are any number of people who are sensitive or have an allergic response to substances used on their faces —face soaps, hair washes, face powders, cold cream, vanishing cream, hormone cream, etc. Not everyone, only about 1 or 2 per cent, may be irritated by them. It is quite possible that you can say, "Well, this sort of thing is due to some general infectious process," but if you take slides and make smears you have to look a long while to find organisms which could have caused the trouble. The focal reaction of many of these people has been at the age or at the time when we took everything loose away from them. So you have to come back now to a very definite local reaction to some chemical substance.

One of our patients had a severe vitamin B deficiency combined with a sensitivity of his conjunctiva, and he had marked circumcorneal infiltration. We treated him with beta irradiation.

Another patient, who happened to be our druggist, some years ago kept bothering me frequently because of his conjunctivitis. As he was a druggist, I did not feel up to studying him for his allergic states. Finally, in desperation we sent him through the Department of Allergy, and they found him to be sensitive to feathers and house dust. By discarding his bed pillows, he was completely relieved. He is still a drugggist. Since that time we have had two other pharmacists who have come through with ocular conditions that we have found to have been due to very simple substances. Always in the back of our minds is "How are we going to find out what causes it?" It is never the whole materia medica. It is usually something that they are using fairly routinely.

One patient came to us with both corneal involvement and a little conjunctivitis. These patients may have a purely ocular conjunctivitis without any involvement of the lids. Any part of the cornea or corneal margin may be involved in a reaction. Usually, however, it is at three or nine o'clock, in the region of the pinguecula. If the eyes are constantly overstimulated, the result will be a pterygium with a corneal overriding.

Most of the cases that we get and treat with beta irradiation have been operated upon once or twice or three times. One was operated upon eleven times as he went from one Army post to another, with a knife-wielding eye surgeon waiting for him at each post. The urge to cut is a very strong one in ophthalmology, and no place are patients hacked more frequently than on these recurring pterygia.

Another pterygium we saw was just beginning to develop. If you do not remove the offending substance, a pterygium will develop that you can cut after a while. But if you get it at this early stage and remove the offending substance, it will retract and give practically no further trouble. You cannot operate on a pterygium and forget it because it will tend to recur due to the repeated stimulation of the offending substance.

The eyes can be scraped for bacteria and one will be found occasionally, but the offending substance must be found.

In about 90 per cent of cases a few simple questions will bring it to light. If the pterygium breaks down and forms an ulcer, the ulcer will be progressive and be very indolent if you do not remove the cause.

I cannot tell you all the causes because they are as numerous as the substances found. As you can perceive, you can treat the patients as we have treated them, and whenever you see the pearly gray luster it means that the pterygium has been stopped. But treatment should not stop there. While we treat the local condition, we also go over the general history of the patient and find out what is behind the condition, and if necessary he goes through the allergy studies. Allergy is not as complicated as we have made it out to be. Between the general medical man and the allergist we have developed a tremendous mysticism about it that is not essential to the diagnosis, nor is it essential to the treatment.

You will see pairs of pterygia in people who are exposed merely to streams of air, as locomotive engineers, farmers, truck drivers, and others who work where the air is not clear. The dust-laden air comes through and purely mechanically stimulates the beginning of a pterygium. Both eyes of one individual who had this condition were treated by beta irradiation in order to get rid of the corneal overgrowth. Whenever you achieve a pearly luster it is an indication that the vascularization has been interfered with.

We had one case which was interesting because of the involvement of the palpebral conjunctiva. Large so-called follicles or granules lay in the undersurface of the lid, large or small according to the amount of irritation. A small pinguecula was treated with beta irradiation and the offending growth removed.

When the entire ocular conjunctiva is involved it makes a little different story. You cannot remove the entire conjunctiva. It does thicken, and it may take prolonged therapy before the patient is made comfortable. If he starts out with a swelling of the mucous membrane and then has a second attack, he is really quite uncomfortable.

A very serious condition is one in which the corneal vascularization tends to encroach upon the pupillary zone. Do not rely too much on beta irradiation, or on any of the other treatments to get rid of all the blood vessels. The superficial blood vessels, yes. Those on the superficial epithelium can be removed. Those that go down deep below Bowman's membrane to the substantia propria do not clear up and these cases act just exactly like other types of interstitial keratitis.

An individual whom we studied some years ago had a superficial punctate keratitis, iritis and retinitis, and there were many things to treat. Dionin had been used, but he had a sensitivity to it and more than one portion of the eye was involved.

A young lady came to us who knew that she could use only certain types of face powder, but she had the habit of borrowing face powder. She had a straight orris root sensitivity. Both eyes were involved; in one eye practically one third of the cornea was involved. The superficial epithelium had been desquamated, and by staining with fluorescein we could see a very large area. The other areas were not quite as extensive. Both of them were very painful and she was hospitalized. By merely eliminating the use of all drugs and substances the eyes cleared up in about twenty-four hours.

A woman who had some small hemorrhagic plaques in the conjunctiva, also had petechial hemorrhages. She had a

food sensitivity. When she went on a food bout, beautiful conjunctival hemorrhages would occur, which under the slit lamp were very small.

Another woman had repeated attacks of conjunctivitis and bronzing of the eye by pigment infiltration as a secondary change. There was some vesiculation. She had marked involvement of the conjunctiva with large isolated follicles on the ocular conjunctiva as well as corneal involvement. This was caused by a face cream.

Another case exhibited a marked bronzing of the circumcorneal area with infiltration of the ocular conjunctiva. Whenever you see edema or fullness around the eye you must consider the case from three points of view. First, that of some chronic edematous process with hypometabolism or hypermetabolism. With chronic edemas you get hypertension, and finally allergic states that are long standing. It is the persistence in the use of the offending substance that produces bronzing and thickening of the lids.

Occasionally corneal desquamation occurs quite similar to that occurring with herpes or with a virus keratitis. One case we saw was not caused by a virus, but was the result of using overnight cream that produced corneal desquamation. The patient felt it on arising in the morning. On staining with fluorescein very fine punctate spots were seen. This condition can become very serious, especially if the patient persists in using the cream.

There may be any number of superficial corneal scars. Corneal involvement is very common, and one or two attacks do not cause any trouble, but there may be diffuse corneal scarring, and it is not dusted off by the use of beta irradiation or contact x-rays. Neither is lamellar keratoplasty the entire answer. You must get rid of the offending process.

A diffuse corneal scar, not unlike the scar you get with an interstitial keratitis may appear. It looks as though it should be in Bowman's membrane and the superficial epithelium. However, there is some involvement of the anterior portion of the substantia propria, and if the condition persists long enough the scar will permeate the cornea through and through. Of course, the accompanying anterior cyclitis or iritis may cause the loss of most of the useful vision. There may be bilateral scars. Operation with corneal transplants may be done, but again unless you find the offending mechanism you cannot guarantee permanent cure.

I hoped to have a report ready this year on the study of about a dozen cases of keratoconus which we are making with our allergist, Dr. George Waldbott of Detroit. Some years ago I was impressed by a man who had an acute keratoconus at the age of 60. He had developed extreme sensitivity. He drank too heavily and smoked too much. He had asthma and migraine, and he was one of the people who wore contact lenses quite well when he was not having an attack of asthma or hay fever. During that period of time he could not bear his contact lenses because he had so much corneal involvement. The corneal edema which occurs in keratoconus is definitely worse during periods of acute allergic states, whether they are due to hay fever or food ingestion or, as in this case, any number of things that would bring on his asthmatic attacks as well as his hay fever. He imbibed too freely one night. I do not know whether he was riding in a double fog, that is, the fog of the night and the fog of his eyes, but on the way home he picked off a safety stanchion and he did not move it. With that we lost this very interesting problem because keratoconus was developing in his other eye and it was developing quite rapidly.

Keratoconus, I think, is a dewdropping of the cornea very similar to a hive one might have elsewhere on the body. It repeats itself, and at each session with this hive the cornea becomes weaker and colder. We have followed some of these cases at least ten years, and by removing the offending substances and putting the patients on vaccine therapy and rest, we have watched them go along without further progress of the keratoconus.

I might say here that you may think plastic operation or corneal transplant for keratoconus is the answer. It is not, for the simple reason that, in the first place, many of these corneas are so badly thinned there is no place to attach the new cornea. In the second place, the patients do not always do well with another foreign substance or foreign material to aggravate their already allergic state. We had a patient quite recently who had an excellent successful transplant, but almost immediately his new cornea began to cone forward. We have here a very interesting problem to which I think we are gradually finding the answer. At least, in the number of cases that we have followed quite carefully, I think we can say that in most instances the keratoconus may be an embryologic defect or a defect in heredity, but in our opinion most of them are promoted by the allergic state of the individual.

DIAGNOSIS AND TREATMENT IN OCULAR ALLERGY

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Most patients who have any of the numerous forms of ocular allergy also have allergic manifestations involving other parts of the body. They frequently exhibit general signs usually associated with an allergic tendency, such as (1) a slow pulse, (2) low blood pressure, (3) spastic gastrointestinal tract, (4) increased blood eosinophilia, and (5) lowered basal metabolism rate.

The methods used in testing for allergies are (1) history; (2) provocative tests; (3) conjunctival tests; (4) patch tests; (5) skin tests, and (6) injection tests.

HISTORY

Frequently the patient has noticed a reaction to a specific food, material, or substance and is able to tell the examiner what it is.

The general history outline can be brief or extensive. The chief complaint must be determined carefully because, of course, therapy should aim to relieve this complaint. The general history is important especially in regard to nasal allergy, cough, asthma, headaches, urticaria, angioneurotic edema, eczema, and contact dermatitis.

There is a distinct hereditary tendency in allergy and a brief family history should be included.

The present illness should include:

Onset
- Date, sudden or gradual
- Residence or occupation at time
- Change of environment
- Change of furniture, clothing, cosmetics, nail polish, etc.
- Unusual circumstances or contacts
- Unusual drugs or food
- Acute infections or other disease
- Endocrine disturbances
- Other manifestation of allergy occurring at the same time
- The pollen season at the time of onset

Course
- Symptoms, constant or intermittent
- Analysis of acute exacerbations or periods of relief
- Vacation, business, pleasure trip which may coincide with exacerbations
- Relation of attacks to:
  - Season
  - Pollens and seasonal foods

Environment
- Materials in bedroom, living room, kitchen, basement, place of business
- Plants, insecticides, dogs, cats
- Neighborhood: grasses, weeds, trees, factories, stables, etc.

Foods and drugs
- Dislikes and disagreements
- Symptoms caused by foods and drugs

Climate
- Temperature, humidity, wind, sunlight, altitude

Physical agents
- Heat, cold, light

Psychosomatic factors

Other factors contributing to the production of attacks

TESTS

Provocative Tests

The suspected material may be employed in the manner in which it is normally used in an attempt to cause symp-

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toms. If this is not successful, then other methods may be employed.

Conjunctival Tests

The conjunctival site for the administration of the antigen offers a considerable safety factor for the patient. A drop of the suspected antigen placed within the lower lid may produce quite marked changes; and if the speed of the changes indicate too great a sensitivity, the unexpended balance of antigen may be flushed out and the reaction relieved before any harm is done.

Patch Tests

The suspected antigen is placed in contact with the unbroken skin about the eyelids and maintained in contact for one to four days depending on the type of antigen used. This is a reliable method but offers two dangers. One is the tendency of the physician to use too much antigen. This may lead to a sensitization of the patient to an antigen to which he otherwise might never become sensitive. The other danger in the method lies in the materials employed. If the antigens are irritating, gritty, or contaminated, an infection may be caused. If the antigen molecule is of such size that it cannot readily penetrate to the desired cell layer, a false negative or a rather indecisive positive is possible. The good feature of the patch test is convenience.

Skin Tests

Although skin tests are of definite diagnostic importance, their interpretation and significance should be properly evaluated. It is important to determine which reactions are definite or specific and which may be false. In the interpretation of skin reactions, one should take into consideration the variability in the reactivity of individual skins. Varying degrees of dermographia are frequently encountered. In children under the age of 10 years, the scratch test should be employed and in adults both scratch and intracutaneous tests may be used.

Injection Tests

The serologic method of testing for allergies will probably supersede all others when it is completely worked out.

Conjunctival Scrape Smear

The conjunctival scrape smear can be of considerable value if it is properly made and interpreted. Using a smear of secretory material gathered after sleep (especially if this material is obtained by the patient) is to be condemned for several reasons. Objections to attempting a diagnostic examination of this discharge material are:

1. Almost invariably they are too thick for single cell examination.

2. Lysozyme present in the ocular secretion causes lysis of many bacteria which can be seen when other methods are used.

3. Degeneration of cells occurs in the overnight period and may proceed so far that the staining characteristics of the cells will be destroyed or distorted.

4. Debris and other artefacts are more likely to be present and may confuse the picture.

5. In allergic conjunctivitis the allergen may sometimes be seen in a fresh conjunctival smear, but in the overnight specimen there is no way to distinguish artefacts from allergens.

6. If the patient is responsible for handling the slide, foreign matter may become incorporated with the secretion, resulting in an inaccurate and possibly harmful conclusion.

7. The purpose of the smear examination is to determine proportion and distribution of major cell types in the conjunctiva. Discharge material does not give an accurate picture of cell types as found in the conjunctiva. Determination of the presence or absence of eosinophils is the only function served by the

examination of discharge material. The percentage of eosinophils and of other cells together with the rise and fall in respective percentages that are found in the course of the disease cannot be followed.

Method of preparing slides. The best smear we have been able to obtain for ophthalmologic purposes is the scrape smear. A satisfactory scraper is not commercially available but may be very easily home made. It consists of a piece of platinum or Nichrome wire (no. 15 Stubbs gauge) about three inches long. The distal half inch of this wire is carefully and gently hammered to a spade-shaped feather edge about three eighths of an inch wide. This feather edge is filed to a very flat curve, almost straight, and smoothed carefully; when properly made, the edge may be drawn over a sensitive conjunctival surface with enough pressure to indent its own thickness with no feeling of sharpness. The proximal end is mounted in any convenient metal handle (a piece of aluminum rod is ideal) and is then ready for use. Resmoothing of the feather edge must be done just prior to flame sterilization each time the scraper is used. This smoothing is most easily done by drawing the instrument over any convenient glass surface two or three times while pressing the edge down under the ball of the finger. The feather edge is a safety measure for the patient; a twitch of the head or of the eyelid will cause the edge to roll back upon itself instead of piercing the conjunctiva. It is customary to anesthetize the conjunctiva before taking scrape smears, but with the scraper made as described, there is so little irritation that anesthesia is not necessary in most cases. The only feeling the patient has following the procedure is a slight grittiness that disappears very quickly. Another advantage of this type of scraper is that the surface is never penetrated deeply enough to cause bleeding, which might complicate the picture.

The technic of using the scraper described is simple. The scraper is flame sterilized just prior to use; this cleans the metal well enough for cells to cling to it quite firmly. An assistant everts both the upper and lower lids, bringing the everted lid margins together. This apposition of lid margins serves to protect the cornea as well as to expose the maximum conjunctival surface. The scraper is drawn over the conjunctival surface with just enough pressure to indent it slightly. Each lid is scraped three times, using only one side of the scraper for each lid. This will supply enough material to give good cellular distribution to two slides, possibly more. The material is transferred to grease-free glass slides by drawing the scraper blade over one half to two thirds of the slide surface, using one slide for each lid. Usually not much is gained by examining the material from each lid separately, but at times it may be desirable and it has seemed a good habit to observe.

After the slides are smeared, they must be promptly stained. We observe a time limit of two hours from obtaining material to staining. All of the polychrome stains on the market are good; however, for convenience and economy, possibly, the best is Hansel's stain.

Examination of slides. The first examination of the stained slide is always done with the low power of the microscope (100 to 150 diameters). At this magnification, the eosinophils are counted in proportion to total cells counted and the percentage recorded. At the same time that this count is being made other cell types are recorded. They may be examined immediately or their approximate position remembered for later study. The primary examination should not take more than

two to three minutes for each properly prepared slide. Percentage figures are desirable as it is much easier to compare percentages than arbitrary systems of plus marks when later serial observations are done. The higher powers of the microscope are utilized to determine the presence of bacteria, peculiarities in individual cells, and other information that may be of value.

It must be mentioned that Wright's stain is not a good bacterial stain, but it serves to determine the shape and grouping (chains, clumps, packets) of any organisms that may be found, and to determine factual habitat, intracellular or extracellular plus any other gross characteristics that may be present.

Correlation between eosinophil percentages and clinical findings may be of interest.

Percentage of eosinophils	Nature of symptoms
0 to 3	None (with recovery from an attack, visible signs only)
3 to 10	Gradually increasing severity in symptoms
10 and above	Usually severe symptoms

After an attack, the eosinophil percentage will at times decrease more rapidly than the abatement of visible signs, and, conversely, at times during an attack, signs occur more rapidly than the increase in eosinophil percentage. The lag is not predictable nor constant even in the same patient. Due to this fact, a scrape smear by itself may not be of too much value but should be part of a series following the patient through his treatment. Along with the eosinophil count, it will be found that individuals having an infectious condition will frequently have an increase of the polymorphonuclear type of cell. If the percentage of increase and decrease of the polymorphonuclear cells is parallel (roughly) with the curve for the eosinophils, it is an indication that the allergy may be bacterial in nature; however, too much stress should not be laid on this as, frequently, a secondary infection follows the primary allergic attack. If more than 1 per cent of the polymorphonuclear type of cell is seen, an infection should be suspected and, if not already present, may show within a relatively short time.

While using the high power of the microscope to look for bacteria, the epithelial cells should be examined for the possible presence of inclusion bodies. The finding of inclusion bodies in the absence of symptoms is extremely rare but has occurred frequently enough to warrant inspecting the cells for them.

With a properly made scrape smear, no erythrocytes should ever be seen on the slide. If they are present, the conjunctiva should be examined to determine the reason for their presence.

While counting cells at low power and examining cells at high power, watch should be kept for material that might represent the allergen or irritant substance. Frequently, the irritant is accompanied by an insoluble substance that may be seen on the slide. Examples that may be cited are talc or insoluble starch fragments that can nearly always be seen when the guilty material is face powder, or the hair scales that can usually be picked up on a slide when an allergic attack from animal hair sensitivity is in progress.

TREATMENT

In chronic allergic conjunctivitis, cortisone used locally in the eye as drops or given systemically causes a greater drop in the eosinophil count on scrape smears from the conjunctiva and gives better

subjective improvement than other therapeutic agents used. Staphylococcus toxoid, hypodermically, is the next most effective agent in causing a drop in the eosinophil count on conjunctival scrape smears. It is extremely important that in infected cases, cortisone should not be used as it acts as a spreading factor, particularly in epidemic keratoconjunctivitis and acute pneumococcus conjunctivitis. We find that in cases where the conjunctival scrape smear does not show at least 3 per cent eosinophils, cortisone is of little value. It should be used only when the scrape smear shows we are dealing with a definite allergic factor.

CORTISONE AND HYDROCORTISONE

In clinical study, Cortisone and Hydrocortisone have no direct effect on microbial agents themselves. They do profoundly affect the body defenses and the body reactions to injury. This effect is favorable in certain instances as in the local manifestations of bacterial allergy; and, unfavorable in other instances as in the dissemination of tubercle bacilli from systemic foci.

In a clinical study, Cortisone and Hydrocortisone administered topically were effective in the rapid control of all marginal ulcers in a series of twenty-three so-called catarrhal ulcers. Twenty of these were secondary to a staphylococcus blepharoconjunctivitis and three secondary to seborrheic blepharoconjunctivitis.

In a clinical study, Cortisone and Hydrocortisone administered topically and Cortisone systemically were effective in the rapid control of uveitis and episcleritis associated with a collagen disease.

DIAGNOSTIC ANTERIOR CHAMBER PUNCTURE

Among important ophthalmologic problems not one has been studied more fully and patiently than that of the aqueous humor. In our study of nongranulomatous uveitis, we have performed many anterior chamber punctures. Our technic followed that of Professor M. Amsler of Zurich, Switzerland, very closely. We used a Bowman-like needle similar to the regular twenty-six gauge needle. It has an extremely sharp arrowhead point and is fitted to a tuberculin syringe. After the well-anesthetized eye is firmly fixed by an assistant, the puncture is performed near the limbus and 0.1 to 0.2 ml. is withdrawn. The operation in our experience is without danger. Of course, every precaution must be taken against possible outside infection.

The following routine procedure is then carried out:

a. One drop is used for the precipitation of total protein.
b. One drop goes in the Fuchs-Rosenthal counting cell chamber.
c. One drop is used for bacteriologic culture.
d. Two or three drops are centrifuged in the capillary tube of the Verry apparatus and provide us with identification of the cellular elements.

In nongranulomatous uveitis, local cortisone is of definite value if the anterior chamber eosinophil count is 4 per cent or more. If the eosinophil count is less than 4 per cent, cortisone has little value.

CHRONIC RECURRENT MARGINAL BLEPHARITIS

Chronic recurrent marginal blepharitis is one of the very common chronic inflammations of the margins of the lids seen in clinics and private practices. It occurs in two forms: nonulcerative and ulcerative. There is always a thick reddening of the lid margins with scaling or crust formation. In most cases where conjunctival smears and lid margin smears were taken, it was found that

there was a bacterial allergy and, frequently, an eosinophil count of greater than 3 per cent. The primary factor is a hypersensitivity to Staphylococcus, while the secondary factors are a seborrheic type of skin and, in the ulcerative type, a secondary infection due to Staphylococcus aureus or albus.

Best results in the treatment of chronic marginal blepharitis are obtained by the use of local antiseptics such as aqueous zephiran 1:5000 with a little adrenalin, or aqueous metaphen 1:5000 with a little adrenalin, used three times a day, boric acid unguentum locally with staphylococcus toxoid and dust and mold injections subcutaneously. Similar treatment without the dust and mold injections is almost as effective. Cortisone 1:4 dilution used locally every two hours gives very satisfactory results.

PHLYCTENULAR KERATOCONJUNCTIVITIS

Phlyctenular keratoconjunctivitis has a characteristic nodular appearance and is an affection occurring mainly in children. It is an allergic response by the corneal and conjunctival epithelium to some endogenous toxin to which they have become sensitized. The primary factor is a hypersensitivity to bacteria, especially Mycobacterium tuberculosis or Streptococcus, while the secondary factor is unhygienic environment or a dietary deficiency.

In phlyctenular keratoconjunctivitis, aqueous cortisone 1:4 dilution used locally in the eyes gives the best results. Local antiseptics with dust and mold injections or staphylococcus toxoid injections give almost as good results as cortisone.

VERNAL CONJUNCTIVITIS

Vernal conjunctivitis is a chronic recurrent bilateral interstitial inflammation of the conjunctiva of seasonal incidence and unknown etiology. It is characterized by flat-topped papules usually on the tarsal conjunctiva resembling cobblestones in appearance and a gelatinous hypertrophy of the limbal conjunctiva, either discrete or confluent, accompanied by corneal involvement. It is associated with itching, redness of the eyes, lacrimation and mucinous discharge usually containing eosinophils. The primary factor is a hypersensitivity to various allergens, especially pollen or contact allergens, while the secondary factor is an endocrine or hormonal deficiency.

In vernal conjunctivitis, aqueous cortisone 1:4 dilution used locally in the eyes gives the best results. Local antiseptics with endocrine therapy and staphylococcus toxoid injections also give very good results. Local antiseptics with endocrine therapy results in improvement in more than half the cases.

OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

LELAND H. PREWITT, M.D.
OTTUMWA, IOWA

MY PRIMARY interest in presenting this paper is to bring to you a clinical evaluation of cases of allergy and the treatment of the patients on that basis, not depending so much upon a multitude of allergy tests but relying more upon a detailed history of allergy. The drugs used primarily are histamine, a specific therapeutic agent, in high dilutions, and staphylococcus toxoid, a nonspecific blocking agent, coupled with an elimination program involving avoidance of sensitizing contacts, inhalants and foods.

Histamine is a specific vasodilator and is effective in high dilutions, routinely 0.1 cc. dilution up to 1:10 million being used. Staphylococcus toxoid prevents a recurrence of the allergic reaction. This drug is often given in doses of one unit or less.

We have been following Hansel's technic[2] of high dilutions, using doses of allergenic material up to 1:10 billion. Derlacki and Shambaugh[1,8] have discussed the use of house dust in dilutions up to 1:10 million and higher.* We are not trying to supplant intravenous histamine therapy but offer this as a suggestion for routine office treatment before hospitalization and surgery are advised.

Allergy is divided into physical allergy, psychosomatic allergy and the antigen-antibody reaction.

A pieceworker who had been working under a great deal of tension for years, while at work suddenly developed an esotropia and also retrobulbar neuritis. His vision was reduced to 20/200. Histamine 1:10 million, 0.1 cc., was administered intradermally every three days. After two weeks he was back on the job, diplopia cleared up and vision returned to 20/20.

Such cases may improve without treatment. I suggest, however, that if they must be treated, one should try the high dilution therapy as advocated by Hansel[2] and many of his followers, before trying hospitalization and surgery.

Another case of retrobulbar neuritis occurred in a mail carrier who complained of loss of vision in the right eye. He was sensitive to paper and glue. He had been a mail carrier all his life. We placed him on paper and glue therapy, along with histamine therapy, and in about three weeks his vision returned.

At the American College of Allergy meeting in Pittsburgh in April 1952, Ruedemann[7] mentioned several cases of retrobulbar neuritis as well as several cases of retinal detachment. One of the 4 cases of retinal detachment which he mentioned was that of a young man who had experienced four detachments in one year due to chocolate. Had this man been operated upon without his allergic contacts being eliminated, the operative end results would probably not have been successful.

I urge you, before carrying out your operative procedure, to take a good history for allergy in all cases. The history of allergy is very important as is the running history that follows with the second, third and later visits. Find out how your patient has been getting along, whether he is better or worse, for on this clinical information will depend the decision to increase or decrease the therapy.

* These solutions in high dilutions can be obtained from the Hollister-Stier Laboratories.

Presented at the meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy, Oct. 17, 1952, Chicago, Ill.

I should like to emphasize that if good results are obtained with 0.1 cc. of 1:10 million dilution, that same dosage should be continued. The tendency is to use a more concentrated dose, but I would urge you to use higher dilutions rather than lower.

We routinely start patients on no. 7 dilution (which is 1:10 million), 0.1 cc. every three to five days. Because so many allergic conditions are caused by occupational duties, it is not easy to eliminate the causative factor. Since it is necessary to keep these patients on the job, one cannot always follow strict elimination therapy. Farmers often become sensitized to rust, smut, molds, animal danders and pollens involving their hands, eyes and ears, and this sensitization often produces asthma.

One young woman was sensitized to house dust and wool. She had multiple recurrences affecting her eyes, oftentimes involving the upper and lower lids, which caused them to swell until they were completely closed. We placed her on house dust therapy in high dilutions to which she responded very nicely. As soon as she returns to her contact, she is affected again, but she knows what her sensitizing allergens are. She comes in when she gets into difficulty and manages without hospitalization, undue expense or time lost from work.

A young man, who drank about a fifth of whiskey a day, had a marked involvement of the eyelids, and he also had a considerable loss of hearing. We urged him to stop his drinking, which he did finally. He made a fairly good recovery, then promptly went back to drinking. We tried to put him on small doses of sedative to stop his tremor, but he said he was afraid he would get the drug habit. He was like the man who lost his hearing because he would rather drink and not hear than to hear and not drink. At the present, we are treating him with histamine, 0.1 cc. of 1:10 million dilution, every five days, plus staphylococcus toxoid in the same dosage.

A woman patient, after operation for cataract, developed blepharitis and beginning corneal opacities. We placed her on staphylococcus toxoid therapy, 0.1 cc. of 1:10 million dilution, every five days. The blepharitis and the corneal opacity improved.

One of our men patients has the pustular type of acne, about as severe a case as I have seen. We placed him on staphylococcus toxoid and eliminated different kinds of food. He gradually made some improvement but he does eat chocolate and foods that he should not. He does not take his trouble too seriously, which results in recurrences.

A high school professor was sensitive to penicillin. He had been using penicillin lozenges, became sensitized, and came in with a very red throat and difficulty in swallowing. He also had considerable involvement of his feet. He had a history of having had athlete's foot. He was placed on one unit of staphylococcus toxoid and 0.1 cc. of 1:10 million dilution of Trichophyton, Monilia and Epidermophyton. His condition showed improvement in two days.

For three years a girl had been treated for marked dermatitis of her knuckles and the back of her hands. They were unsightly, which presented quite a social problem. Along with that, she had a marked blepharitis and a superficial punctate keratitis. Her eyes bothered her all the time. She worked in the packing department of a large manufacturing plant where there was considerable sodium silicate glue and paper dust. Waldbott[9] points out that each hand lesion is due to a certain pattern of contact and he tries to work out the causative agent by the localization of the lesion. We tried to find out whether boxes bumped her knuckles, but were unsuccessful. We did learn that in combing her hair in the evening, she ran her knuckles through her hair. So we suggested that she wear gloves on her hands and goggles over her eyes while combing her hair, and a cap during work. She could not quit her work. We placed her on paper extract, staphylococcus toxoid and house dust. She has been able to return to work and has made a complete recovery.

Severe facial neuralgia occurred in a girl typist 28 years old. We tried 0.1 cc. doses of histamine, 1:10 million dilution, which gave excellent results. I have had three or four such patients, one of whom threatened to commit suicide because the pain was so severe. The neuralgia cases are difficult to treat, as they tend to recur and do not respond as well as do the cases of histaminic cephalalgia.

A striking example of the response to treatment of a case of histaminic cephalalgia was that of a white man, 50 years old, who had had severe attacks of pain for two years over the right side of the face, radiating to the right eye and nose, causing tearing and congestion

of the right side of the nose. These symptoms occurred most often at night and were so severe that he got out of bed and walked to relieve the pain. He was put on 0.1 cc. of histamine, 1:10 million dilution, every three days for five treatments, which cleared up his symptoms entirely. Such cases respond to histamine therapy quickly, perhaps because the prodromal period of vasodilatation is short, as these attacks come on suddenly.

For two years a woman patient had been treated for dermatitis of the face, which remained red all the time. She showed sensitivity to house dust and to corn. We put her on an elimination diet, house dust and staphylococcus toxoid, with immediate improvement.

I might mention that staphylococcus toxoid is comparable to cortisone. Frequently one will get a reaction in diabetic patients very similar to the reaction from cortisone and the oxysteroids.

Another woman, 45 years old, had a submaxillary swelling. Smears of the saliva showed eosinophils. She chewed gum continually and ate a great deal of popcorn. On eliminating both and by using histamine, the swelling cleared up. She has had no further difficulty up to the present time.

One patient had had a thyroidectomy two years previously with a resulting keloidlike formation. The scar was constantly red and inflamed and had been treated with ointments of various kinds. We used nothing but staphylococcus toxoid no. 7. The tendency toward keloid formation in most cases is due to autonomic dysfunction with arteriolar contraction and venule and capillary dilatation, with anoxia and an accumulation of fibrous tissue. This case responded very well and the inflamed area cleared up completely.

Allergy histories will reveal what food is eaten. Some of these patients eat the same food day after day. *Food Allergy* by Rinkel and associates[6] gives suggestions as to food allergies. We have treated a psoriasis involving the ears, external canals and scalp, and several cases have responded very well. We also use 10 per cent solution of silver nitrate on the lesions.

In cases of extreme hypersensitivity one must use higher dilutions than are normally used, 1:10 billion or higher. The amount of therapy to be used is determined by the history, the degree of symptomatology and the skin tests. As Derlacki and Shambaugh[1] mentioned, many of these cases exhibit no reaction to the skin tests; therefore, they are of no value. The history is much more important. A very careful detailed history covering seven days a week, twenty-four hours of the day will often reveal the answer.

Skin tests are not an absolute necessity. The therapy advocated here will give very good results. I have been studying allergies for twenty years and have known of Hansel's work[2] for the same length of time. We used to use concentrated doses of histamine and staphylococcus toxoid. We gave an injection and then ran for adrenalin and coramine and spent time resuscitating the patient, especially the older individuals. As indicated here, one should use the high dilution therapy. With concentrated dilutions one will precipitate an attack instead of preventing it. The average dilution used here is 1:10 million, 0.1 cc. intradermally. I would stress, *intradermally*. I know some men disagree, but as Spector says, the neurocrest cells are epidermal in origin, and by intradermal injections better results are obtained.

Table I based on the severity of the patient's symptoms will serve as a guide for histamine therapy. The dosage is regulated on a clinical evaluation rather than on a series of skin tests.

The technic for dilutions for histamine, house dust, molds and other allergens is indicated in table II.

Staphylococcus toxoid comes in 1,000 units per cubic centimeter and in 1 to 100 units per cubic centimeter (table

TABLE I

CLINICAL CHART AND DOSAGE SCHEDULE

1:100,000	1:1,000,000	1:10,000,000
Very mild nasal allergy, stuffy type, postnasal drip	Moderate, mild, stuffy, postnasal discharge. Occasional sneezing	Moderately marked obstruction, sneezing and discharge. Symptoms may be intermittent
1:100,000,000	**1:1,000,000,000**	**1:10,000,000,000**
Marked sneezing, obstruction and discharge, itching, continuous symptoms, marked nasal changes	Very severe, same as previous group. Marked edema. Polyps edematous	Very marked asthma. Small children with nasal allergy or asthma

III). We use the 1 to 100 units per cubic centimeter, diluting 1 cubic centimeter with 9 cubic centimeters of normal saline solution, so that 0.1 cc. contains 1 unit.[3-5]

The therapeutic suggestions for staphylococcus toxoid therapy are as follows: angioedema, penicillin reaction, staphylococcus reaction and infection, contact keratitis and blepharitis. For children with infantile eczema, rheumatism and arthritic conditions, use 0.10 unit. As mentioned previously, staphylococcus toxoid acts as a nonspecific blocking agent in allergic reactions.

SUMMARY

The clinical features and possible pathogenesis of different allergy entities have been discussed on a clinical basis rather than on multiple skin tests. It is our belief that treatment can be instituted on symptomatology using Hansel's technic[2] of high dilution therapy.

Emotional and psychosomatic therapy, food elimination, house dust, pathogenic molds, vasodilators, resins and other forms of therapy, specific and nonspecific, often have a synergistic action with histamine and staphylococcus and aid a great deal in cases which resist other forms of therapy.

TABLE II

TECHNIC OF DILUTIONS FOR HISTAMINE, HOUSE DUST, MOLDS OR OTHER ALLERGENS

Histamine acid phosphate, 2.75 mg. in 1 cc. ampule containing 1 mg. per cubic centimeter, represents 1 cc. of 1,000 dilution or No. 3. Begin with dilution No. 4.

DILUTION CHART

	Cubic Centimeters		*Cubic Centimeters*		
No. 1	*0.20*	*Allergenic Extract*	*1.80*	*Diluent 2*	*1:10*
No. 2	0.20	1:10	1.80	2	1:100
No. 3	0.50	1:100	4.50	5	1:1,000
No. 4	1.00	1:1,000	9.00	10	1:10,000
No. 5	1.00	1:10,000	9.00	10	1:100,000
No. 6	1.00	1:100,000	9.00	10	1:1,000,000
No. 7	1.00	1:1,000,000	9.00	10	1:10,000,000
No. 8	1.00	1:10,000,000	9.00	10	1:100,000,000
No. 9	1.00	1:100,000,000	9.00	10	1:1,000,000,000
No. 10	0.50	1:1,000,000,000	4.50	5	1:10,000,000,000

TABLE III

STAPHYLOCOCCUS TOXOID CLINICAL CHART AND DOSAGE SCHEDULE

Ten units per cc.	One unit per cc.
Urticaria, angioedema, penicillin, staphylococcus infections, contact dermatitis	Infantile eczema, rheumatism, arthritis
1.0 unit 0.10 cc. 0.80 unit 0.08 cc. 0.50 unit 0.05 cc. 0.30 unit 0.03 cc. 0.20 unit 0.02 cc.	0.10 unit 0.10 cc. 0.05 unit 0.05 cc. 0.03 unit 0.03 cc. 0.02 unit 0.02 cc.

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NEW INSTRUMENTS AND NEW TECHNICS

If you have a new instrument or a new technic which you wish to report at the annual meeting of the Academy in October, please notify the Secretary in charge of your Scientific Section as soon as possible. The deadline is September 15. It will not be possible, as formerly, to make arrangements during the meeting for such presentations.

The Secretary for Ophthalmology is Dr. Kenneth L. Roper, 55 East Washington Street, Chicago 2. Dr. Howard P. House, 1136 West Sixth Street, Los Angeles 17, is Secretary for Otolaryngology.

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Editorials

AUDIOVISUAL AIDS

FOR nearly twenty years the Academy has utilized motion pictures in the scientific program and in the instruction courses at the annual convention. In the beginning the original films used were black and white, 35 millimeters, made by amateur photographers (often the surgeon, for they were mostly on surgical procedures), with variable lighting and variable backgrounds. They were inexpensive and easy to show. They were very popular and soon special provision had to be made for their exhibition. At one time the Council appropriated $500 for the production of a film on eye surgery, but voluntary productions filled the program and the funds were never used.

The development of a ciné program has been along two lines: to document scientific procedures and to demonstrate technical procedures. The field of usefulness has become wider with improved technics in photography and with more carefully worked out plots and storyboards. The film on Embryology of the Eye, produced by the Academy, was a pioneer that has been approved the world over for medical school film libraries.

A companion film on Embryology of the Human Ear is currently in production, and the first part probably will be available for exhibition at the October meeting of the Academy. In the preparation of this film it was necessary to make special preparations to fill gaps in the knowledge of the origin and development of certain parts of the ear and to correlate certain known facts with reference to precedence and coincidence. The Carnegie Institution, Department of Embryology, in Baltimore, under the supervision of Dr. George Corner, again, as in the film on Embryology of the Eye, gave generously of assistants and time for the working out of these problems. Until the film was under way, the serious gaps in knowledge of the embryology of the ear were not generally recognized. The picture will go a long way toward clearing up obscure points in anatomy.

Motion pictures are particularly useful in teaching training technics by repetition. Certain surgical procedures can be shown by exhibition of a few feet of film, discussed and shown again and again. Examples of this repetitive method of teaching will be exhibited in the courses on instruction. A pilot showing

will demonstrate the McLean cataract suturing technic. The picture will be on a loop film that runs continuously, repeating the full picture every few minutes. Teachers and students are requested to see this demonstration of a new method of using motion picture film in class rooms. The demonstration is made by Sturgis-Grant Productions, Inc., producers of the film on Embryology of the Human Ear, with equipment loaned by Seminar Films, Inc., New York, N. Y.

W. L. BENEDICT, M.D.

ATLASES

WHEN the Academy undertook the distribution of the Atlases of Ophthalmic Pathology and Otolaryngic Pathology, the Instruction Courses (1921) and the Home Study Courses (1940) were already established with definite objectives and proven plans of operation. Pathology was no longer static, and the Army Medical Museum was the depository of thousands of specimens from military and nonmilitary sources that had been carefully studied and cataloged at the request of and with the financial assistance of the Academy. Collections of histopathological sections for study were made available to students, particularly residents in hospital training and candidates for Board certification, and many sets were placed in schools for class work. The logical development of the teaching program in pathology was the publication of an atlas with description of each slide and comments made by the staff pathologist. The atlases produced by the Army Medical Museum (now the Armed Forces Institute of Pathology) went through three editions with evident improvement in each succeeding revision.

The atlases, however, were not geared to any textbook of pathology and the student was on his own. Teachers and students realized the necessity of correlating the pictures with a modern conception of physiologic pathology, and when the opportunity came to produce an atlas from the abundant pathological material in the Armed Forces Institute of Pathology, combined with a text on ophthalmic pathology by Dr. Jonas Friedenwald, the Academy underwrote the production cost and sponsored the fourth revised edition. (Government restrictions at that time prohibited the sale of the atlases by the A.F.I.P. and the Tumor Registry.)

The Atlas of Ophthalmic Pathology was introduced at the Academy meeting in 1952 and has been universally acclaimed as one of the outstanding books of its kind. Negotiations are now under way for a Spanish edition.

The Atlas of Otolaryngic Pathology took longer to compile. Interruptions in the services of personnel at the Armed Forces Institute of Pathology were partly responsible, but the important thing for the Atlas was the preparation of a huge mass of new material and the selection of the best slides for teaching purposes. Now the nearly completed Atlas of Otolaryngic Pathology is on the press and, we hope, will be available at the October meeting. The completion of these atlases of pathology marks an epoch in the educational program of the Academy comparable to the completion of two basic films on embryology.

What is the next big project? An atlas of drawings and good photographs of the chamber angle, an Atlas of Gonioscopy, is sorely needed to supplement the advanced studies in glaucoma. Some pictures have been published to illustrate specific features, others as examples of the engraver's art to reproduce spectacular innovations in anatomical revelations regardless of their clinical significance. These pictures are scattered in the literature of Ophthalmology, in books and magazines and adver-

tising media, a hodgepodge of art with some distant relation to usefulness in medical science.

The production of an Atlas of Gonioscopy is possible. The deterrent is the original cost. The members of the Academy can best decide on the advisability of subsidizing and sponsoring such an aid to advanced teaching.

W. L. Benedict, M.D.

HOME STUDY COURSES

The Home Study Courses offered by the Academy are recommended by the American Boards to their candidates; they are widely used as an adjunct to residency training programs; and they are a valuable aid to clinicians as a review of the basic sciences or as an outline for a systematic study program.

The brief quotations following are representative comments from physicians in various stages of training for the two specialties and those already in clinical practice.

This month I have completed the enjoyable experience of taking your Home Study Course in Ophthalmology. I should like to congratulate you on the amazingly excellent organization of this Course, and also upon the wide scope of its content.

*

I wish to thank those responsible for the organization and administration of the Home Study Course and the faculty. To people like myself, who have been away from prolonged academic contact, this method of study is most enjoyable and helpful.

*

This Course is an excellent opportunity for one in the service (military) or out of the service. Some of our men here with whom I have spoken, in other branches of medicine, wished the other societies made available similar opportunities for study. I wish to sincerely thank you people who have participated in giving beginners this opportunity for advancement.

*

I wish to take this opportunity to express my thanks and appreciation for all that the Home Study Course has done for me. It has been very stimulating and many times worth the hours expended.

*

I have thoroughly enjoyed this Course and believe it will be of great benefit to me in the future.

*

I want to take this opportunity to thank you for the privilege of taking the Home Study Course. I feel I got a lot out of it. Those responsible for the Course are to be congratulated for giving their time and efforts to help others.

Registration for the 1953-1954 Home Study Courses must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the Executive Secretary-Treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

Letter to Editor

Headquarters,
USAF School of Aviation Medicine
Randolph Air Force Base
Randolph Field, Texas

8 October 1952

Dear Dr. Benedict:

Dr. Hedwig S. Kuhn, Secretary of the Joint Committee on Industrial Ophthalmology, has asked if I would write you to clarify the status of the contract on ocular burns which we have with Tulane University. This is contract No. AF 18 (600)-304. Its title is "Ocular Burns." It became effective on 15 May 1952, and is scheduled to run for a three-year period. Its purpose is:

a. The contractor will conduct a series of scientific investigations on wound repair of the burned eye and related problems; on the site and mode of development of primary and secondary damage in ocular burns; and on the effects of various drugs, other agents, and medical and surgical procedures on the healing of eye burns.

b. The contractor will undertake to resolve currently controversial research and clinical findings of fundamental importance in the ocular burn problem.

c. The contractor's investigations will include studies of cell damage; cell division; effects of various normal and abnormal factors on cell functions; effects of drugs, hormones, other compounds, and cellular degradation products on healing; buffer solutions for neutralizing injurious agents; iontophoretic withdrawal of injurious agents; and the optimum materials and procedures for grafting in the treatment of eye burns.

The principal investigator is Dr. James H. Allen of the Department of Ophthalmology, Tulane. The research is to be a large scale investigation of this problem. The project is under way at the present time and we have already had one preliminary report on its progress. Since there seemed to be some misunderstanding about the exact status of this contract, I believe the above will clarify it.

Yours sincerely,

VICTOR A. BYRNES
Colonel, USAF (MC)

In Memoriam

Ralph Almour.....New York, New York
March 27, 1953

Gordon Berry.....Worcester, Massachusetts
March 17, 1953

Benton Noble Colver.....Los Angeles, California
March 14, 1953

Thomas Carroll Davis.....Philadelphia, Pennsylvania
March 5, 1953

William Carle Davis.....Columbus, Ohio
March 8, 1953

John Norris Evans.....Brooklyn, New York
April 9, 1953

John Frank Hardesty.....St. Louis, Missouri
June 21, 1953

Will Wanee Holmes.....Logansport, Indiana
April 28, 1953

Andrew Joseph Lamb.....Cleveland, Ohio
February 9, 1953

Hugo Reim.....St. Louis, Missouri
April 15, 1953

George Hocken Thompson.....Pittsfield, Massachusetts
March 8, 1953

FINANCIAL REPORT

Annual Audit for Calendar Year — 1952

April 21, 1953

The American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

As requested, we have examined the balance sheet of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, Rochester, Minnesota, for the year ended December 31, 1952, and the related statements of operations and net worth for the year then ended. Our examination was made in accordance with generally accepted auditing standards, and included such tests of the accounting records and such other auditing procedures as we deemed necessary in the circumstances. We submit herewith our report, which includes the following statements:

Exhibit

"A"—Balance Sheet, December 31, 1952
"B"—Statement of Operations, Year Ended December 31, 1952
"C"—Analysis of Undisbursed Funds from Special Contributions
"D"—Analysis of Net Worth Account
"E"—Analysis of Increase in Market Value of Securities

Schedule

1—Securities Owned, December 31, 1952

FINANCIAL CONDITION

A balance sheet as at December 31, 1952, appears in Exhibit "A." Subject to the comments contained in this report, we believe that this statement correctly reflects the financial condition of the American Academy of Ophthalmology and Otolaryngology on December 31, 1952.

The following comments describe in more detail the nature of the various assets and liabilities and the extent of our verification.

Current Assets, $20,602.69

Cash on deposit at the various banks, totaling $3,434.53, was verified by communicating with the depositories, the funds in the Canadian bank being stated in U. S. dollars. Petty cash fund of $25.00 was verified by actual count during the course of our examination.

Membership dues receivable, in the amount of $1,044.00, consists of delinquent dues for 1952 and prior years which were verified from the records only. Other receivables totaling $997.18 are amounts due from members and others for services, films, slides, abstracts, manuals, etc.

The inventory of abstracts, manuals, bound volumes, films and slides, in the amount of $15,101.98, was taken from the inventory records without further verification except as to tests of prices, extensions and footings.

Investments, $231,546.25

In Schedule No. 1, we present a schedule of bonds and stocks which had a market value of $231,546.25 on December 31, 1952, as compared with $242,854.51 on December 31, 1951. U. S. Savings Series G Bonds are stated at maturity values rather than current redemption prices.

Securities in the "Educational Fund," which are held for safekeeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, were verified by communicating with the custodian of these securities. U. S. Savings Series G Bonds in the "Wherry Memorial Fund" are held by Dr. W. L. Benedict, Executive Secretary-Treasurer, and were inspected by us. Interest earned on bonds since the dates on which last payments became due has not been accrued. An analysis of the increase in the market value during the year is shown in Exhibit "E."

Fixed Assets, $32,826.58

Furniture, fixtures and equipment amounting to $8,925.58 represent equipment acquired by the Academy over a period of years. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried and appraised at a market value of $1,420.28 as of January 1, 1944, and set up on the books for that amount. Equipment purchased, less exchanges, since September 1, 1942, amounting to $7,505.30, was for the most part verified through an inspection of supporting vouchers, and of the latter amount $549.75 was purchased during the current year. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Embryology motion picture film and slides, in the amount of $40,900.00, represents the cost to complete a medical motion picture film, "Embryology of the Eye," and slides, by the

Sturgis-Grant Productions, Inc. The master film is being used to produce prints and slides and these are sold to those requesting them. The cost of the original film and slides is being amortized at a rate that appears to be adequate.

Deferred Charges, $22,354.61

Unexpired insurance premiums as computed by us amounted to $212.58. Supplies in the amount of $286.65 represents stationery stock on hand as computed by employees of the Academy without further verification.

Prepaid postage deposit of $188.71, verified by correspondence, represents the balance of cash advanced to the U. S. Post Office at Omaha, Nebraska, to be applied as postage on subsequent mailings of the TRANSACTIONS and other periodicals by the publisher at Omaha.

Advance payments, totaling $21,666.67, have been made to Sturgis-Grant Productions, Inc., to produce a film, "The Embryology of the Human Ear." Of this amount, $7,500.00 represents the cost of producing "script and storyboard" and the balance of $14,166.67 is the initial one-third cash advance on a contract dated July 10, 1952, for the production of the film, which will cost $42,500.00. The balance of the contract, $28,333.33, which is not shown on the balance sheet as a liability, is to be paid in two installments: $14,166.67 when the film reaches 50 per cent of completion, and the balance of $14,166.66 on delivery of film.

Current Liabilities, $12,841.69

Accrued expenses of $7,747.24 represents unpaid December printing cost of the TRANSACTIONS and other current expenses. Membership dues, etc., totaling $227.95, represents amounts paid in advance by association members and others. Advance payments of $2,420.00 for bound volumes consists of payments received as of December 31, 1952, for which volumes have not as yet been delivered. Withholding tax payable amounted to $646.50, while notes payable of $1,800.00 represents a temporary loan which was verified by correspondence.

Undisbursed Funds from Special Contributions, $10,269.68

Undisbursed funds from contributions received and designated for "Specific Projects" totaling $10,269.68 are analyzed in Exhibit "C," which shows 1952 transactions as well as prior year balances.

Net Worth, $284,218.76

An analysis of the net worth of the Academy is shown in Exhibit "D." The loss from current operations was greater than the increase in market value of securities, resulting in a decrease in net worth from $298,138.35 on December 31, 1951 to $284,218.76 on December 31, 1952.

OPERATIONS

A statement of operations for the year ended December 31, 1952, is presented in Exhibit "B." A summary and a comparison with the results for the year 1951 is as follows:

	Year 1952	*Year* 1951	*Increase* **Decrease**
Revenues	$193,370.79	$172,681.10	$20,689.69
Operating expenses	210,782.89	173,558.72	37,224.17
Net Loss	$ **17,412.10**	$ **877.62**	$16,534.48

The increase in net loss from a year ago is due principally to increased cost of publishing the TRANSACTIONS and convention expenses.

GENERAL

Upon examination of the insurance policies in force, we found fire and extended coverage on furniture, fixtures and equipment in the amount of $7,500.00. Paper stock, publications, etc., in custody of the publisher at Omaha, Nebraska, are insured for $12,000.00. Comprehensive general liability insurance in force is in the amount of $25,000.00 for each accident, with a total limit of $25,000.00. Special limits of $50,000.00 for each accident, and a total limit of $100,000.00 cover the Chicago convention activities with property damage limits of $5,000.00. Workmen's compensation insurance, which is being carried at the statutory amounts, now includes coverage for all persons employed at your annual convention.

The Executive Secretary-Treasurer is bonded for $25,000.00, while the principal office employees are each bonded for only $1,000.00. This Fidelity Position Bond coverage for office employees, in our opinion, is inadequate. Office cash and securities are covered up to $2,400.00 within the premises and $2,100.00 outside the premises against safe burglary and messenger robbery. During convention time the coverage of office cash and securities on the premises is increased to $18,000.00 with $22,000.00 for securities only.

We acknowledge the very courteous assistance extended to us by Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, and his staff during the progress of this examination.

OPINION

In our opinion, subject to the comments in the preceding paragraphs, the accompanying balance sheet and related statements of operations and net worth present fairly the financial position of the American Academy of Ophthalmology and Otolaryngology at December 31, 1952, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,
BYERS, WOBSCHALL & MILLER
Certified Public Accountants
By Edw. A. Wobschall, C.P.A.

EXHIBIT "A"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Balance Sheet as at December 31, 1952

ASSETS

Current Assets:			
Cash, First National Bank, Rochester, Minnesota		$ 77.22	
Cash, First National Bank, Minneapolis, Minnesota		1,249.44	
Cash, Bank of Montreal, Toronto, Canada		2,107.87	
Petty cash fund		25.00	
Membership dues receivable, delinquent		1,044.00	
Accounts receivable, others		997.18	
Inventory, abstracts, manuals, monographs, films, etc		15,101.98	
Total current assets			$ 20,602.69
Investments: (Schedule No. 1)			
Bonds and stocks			231,546.25
Fixed Assets:			
Furniture and fixtures	$ 8,925.58		
Less reserve for depreciation	5,149.00	3,776.58	
Embryology of the Eye, Film & Slides	40,900.00		
Less reserve for depreciation	11,850.00	29,050.00	32,826.58
Deferred Charges:			
Prepaid insurance		212.58	
Supplies		286.65	
Prepaid postage		188.71	
Embryology of the Human Ear Motion Picture Film		21,666.67	22,354.61
Total Assets			$307,330.13

LIABILITIES AND NET WORTH

Current Liabilities:		
Accrued expenses	$ 7,747.24	
Deferred income, membership dues, etc.	227.95	
Deferred income, advance payments on bound volumes	2,420.00	
Withholding tax payable	646.50	
Notes payable	1,800.00	$ 12,841.69
Undisbursed Funds from Special Contributions: (Exhibit "C")		
Wherry Memorial Fund	5,177.72	
Sub-Committee on Noise in Industry	5,066.96	
Isabelle Wilson Research & Education Fund	25.00	10,269.68
Net Worth (Exhibit "D")		284,218.76
Total Liabilities and Net Worth *		$307,330.13

* Subject to report comments

EXHIBIT "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Operations

Year Ended December 31, 1952

Revenues:			
Membership dues			$ 30,727.00
Membership applications			7,740.00
Abstracts, manuals and monographs			9,544.60
Certificates			1,894.00
TRANSACTIONS			46,421.65
Home Study Courses			6,718.00
Convention			78,531.50
Testing and calibration of tonometers			1,304.00
Films and slides			2,450.00
Income from securities:			
Interest, Educational Fund		$ 4,713.86	
Dividends, Educational Fund		2,468.15	7,182.01
Other Income			858.03
Total Revenues			193,370.79
Operating Expenses:			
General administrative		72,913.52	
TRANSACTIONS		74,934.96	
Home Study Courses		7,867.78	
Convention expenses		49,983.15	
Educational fund:			
Supplements, etc.	$ 646.61		
Depreciation, film and slides	4,090.00	4,736.61	
Total Operating Expenses			210,436.02
Operating Loss			**17,065.23**
Other Expenses:			
Loss on sale of securities			346.87
Net Loss			$ **17,412.10**

EXHIBIT "C"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Undisbursed Funds from Special Contributions

WHERRY MEMORIAL FUND		
Balance, December 31, 1951		$ 5,012.72
Receipts:		
Interest on U. S. Savings Bonds, year 1952	$115.00	
Contributions received	50.00	165.00
Balance, December 31, 1952		$ 5,177.72
ECONOMIC SURVEY OF IMPAIRED HEARING FUND		
Balance, December 31, 1951		$ 957.46
Receipts: Contributions received		300.00
Total		1,257.46
Less: Expenditures during 1952		1,257.46
Balance, December 31, 1952		- 0 -
SUB-COMMITTEE ON NOISE IN INDUSTRY FUND		
Balance, December 31, 1951		$ 2,429.87
Receipts: Contributions received in 1952:		
Committee on Conservation of Hearing (Transfer)		5,066.96
Other outside sources		3,750.00
Total		11,246.83
Less: Expenditures during 1952		6,179.87
Balance, December 31, 1952		$ 5,066.96
ISABELLE WILSON RESEARCH & EDUCATION FUND		
Balance, December 31, 1951		$ 25.00
Receipts and disbursements in 1952		- 0 -
Balance, December 31, 1952		$ 25.00

EXHIBIT "D"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Net Worth Account

For Year Ended December 31, 1952

Net Worth, December 31, 1951		$298,138.35
Additions:		
Increase in market value of securities during 1952 (Exhibit "E")		3,492.51
		301,630.86
Deductions:		
Net loss, year ended December 31, 1952 (Exhibit "B")		17,412.10
Net Worth, December 31, 1952		$284,218.76

EXHIBIT "E"

Analysis of Increase in Market Value of Securities

	Cost	*Market Value*	*Increase* **Decrease**
December 31, 1951	$241,238.48	$242,854.51	$ 1,616.03
December 31, 1952	226,437.71	231,546.25	5,108.54
Difference	**$14,800.77**	**$ 11,308.26**	$ 3,492.51

SCHEDULE No. 1

American Academy of Ophthalmology and Otolaryngology
Securities Owned, December 31, 1952

EDUCATIONAL FUND

BONDS	RATE %	MATURITY	PAR VALUE	COST	MARKET VALUE *
Citizens Building Co. of Cleveland, 1st Leasehold Sinking Fund	6	1/ 1/46	$ 5,000.00	$ 5,000.00 (1)	None
Van Swerigen Co. Certificates of Indebtedness, Series 1925	6	12/31/48	6,000.00	3,477.03	$ 810.00
Dominion of Canada, 8th Victory Loan Ser. P5	3	10/ 1/63	2,500.00	2,500.00 (2)	2,412.50
Dominion of Canada, 8th Victory Loan Ser. P5	3	10/ 1/63	5,000.00	5,112.50 (2)	4,825.00
U.S.A. Savings, Series F	—	12/ 1/55	13,500.00	9,990.00	12,150.00
U.S.A. Savings, Series G	2½	8/ 1/53	10,000.00	10,000.00 (3)	10,000.00
U.S.A. Savings, Series G	2½	6/ 1/55	4,000.00	4,000.00 (3)	4,000.00
U.S.A. Savings, Series G	2½	3/ 1/56	6,000.00	6,000.00 (3)	6,000.00
U.S.A. Savings, Series G	2½	5/ 1/56	8,000.00	8,000.00 (3)	8,000.00
U.S.A. Savings, Series G	2½	7/ 1/56	2,000.00	2,000.00 (3)	2,000.00
U.S.A. Savings, Series G	2½	11/ 1/56	25,000.00	25,000.00 (3)	25,000.00
U.S.A. Savings, Series G	2½	1/ 1/57	2,000.00	2,000.00 (3)	2,000.00
U.S.A. Savings, Series G	2½	7/ 1/58	30,000.00	30,000.00 (3)	30,000.00
U.S.A. Savings, Series G	2½	12/ 1/58	25,000.00	25,000.00 (3)	25,000.00
U.S.A. Savings, Series G	2½	3/ 1/59	15,000.00	15,000.00 (3)	15,000.00
U.S.A. Savings, Series G	2½	7/ 1/60	30,000.00	30,000.00 (3)	30,000.00
U. S. Treasury, 1966-71	2½	3/15/71	10,000.00	10,087.50	9,568.75
Total Bonds				$193,167.03	$186,766.25

STOCKS	KIND	PAR VALUE	SHARES		
Citizens Building Co. of Cleveland	Shares	No Par	50	None	None
Commonwealth Edison Co.	Common	$25.00	100	$ 2,619.00	$ 3,400.00
E. I. DuPont De Nemours & Co.	Common	5.00	60	2,347.31	5,760.00
Louisville Gas & Electric Co.	5% Preferred	25.00	200	5,450.00	5,900.00
Massachusetts Investors Trust	Trust Cert.	1.00	1,251	18,254.37	25,020.00
Ophthalmic Publishing Co.	Capital	20.00	5	None	100.00
				$ 28,670.68	$ 40,180.00
Total Educational Fund				$221,837.71	$226,946.25

WHERRY MEMORIAL FUND	RATE	MATURITY	PAR VALUE		
U.S.A. Savings, Series G	2½%	1955	$4,600.00	$ 4,600.00	$ 4,600.00
Total Investments				$226,437.71	$231,546.25

* Except for U.S.A. Savings, Series G Bonds, which are included at maturity values
(1) No market available
(2) U. S. dollars
(3) Maturity values

SECTION ON INSTRUCTION

FIFTY-EIGHTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 11-16, 1953
PALMER HOUSE
Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1953 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 9.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to October 1, send orders to 100 First Avenue Building, Rochester, Minnesota; after October 1, mail orders direct to W. L. Benedict, M.D., c/o Palmer House, Chicago, Illinois. Advance orders will not be filled if received after October 8.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not pos-

sible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

CHANGES IN INSTRUCTION COURSE PROGRAM AS LISTED IN THE MARCH-APRIL 1953 TRANSACTIONS

The title of Course 501, to be presented by Dr. Albert P. Seltzer on T-3 and W-3, will be "Plastic Surgery of the Nose, Abrasion of the Face, and Face-Lift Operation: New Concepts and Technics."

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Allen, J. H., et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS M-5, M-6
3	Bedell	Medical Ophthalmoscopy	4 PERIODS M-5, T-5, W-5, Th-5
4	Berliner	Slit Lamp Microscopy: Advanced	4 PERIODS M-4, T-4, W-4, Th-4
5	Birge, et al	New Theories of Vascular Disease	3 PERIODS M-4, T-4, W-4
6	Boeder	Aniseikonia & Space Eikonometry	2 PERIODS T-5, T-6
7	Brown, et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
8	Byrnes	Ophthalmic Filters	2 PERIODS M-5, M-6
9	Callahan, et al	Fundamentals of Plastic Surgery	6 PERIODS M-5, M-6, T-5, T-6 W-5, W-6
10	Clark	Surgery of Ocular Muscles	2 PERIODS T-5, T-6
11 or 12	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
13	Costenbader, et al	Comitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
14	DeVoe	Clinical Application of Gonioscopy	2 PERIODS W-4, W-5
15	Dunnington & Clark	Retinal Detachment	3 PERIODS T-4, W-4, Th-4
16	Fink	Management of Oblique Muscle Problems	3 PERIODS M-5, T-5, W-5
17	Friedenwald	Studies in Aqueous Flow	2 PERIODS M-4, M-5
18	Guibor	Orthoptics: Office & Home Technics	3 PERIODS T-6, W-6, Th-6
19	Hardy, et al	Basic Principles of Orthoptics	4 PERIODS M-4, T-4, W-4, Th-4
20	Henderson	Practical Perimetry	4 PERIODS M-4, M-5, T-4, T-5
21	Hughes, et al	Plastic Surgery: Advanced	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
22	Jardon	Fitting of Ocular Prostheses	2 PERIODS T-5, T-6
23 or 24	Johnson, L. V. & Ballintine	Aqueous Tonography	2 PERIODS M-4, M-5 or T-4, T-5
25 or 26	Johnson, V. C., & Dunlap	Diagnostic Value of X-ray	2 PERIODS M-5, M-6 or T-5, T-6

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
27	Knighton, et al	Highlights of Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
28	Kuhn	Industrial Ophthalmology	3 PERIODS M-4, T-4, W-4
29	Lloyd	Macular Lesions	2 PERIODS M-5, T-5
30	Lowrey, et al	Military Ophthalmology	2 PERIODS M-4, M-5
31	McLean, et al	Cataract Surgery	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
32	Meek	Surgical Anatomy of Orbit	2 PERIODS Th-4, Th-5
33	Meyer, et al	Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
34	Ogle	Essential Aspects of Binocular Vision	2 PERIODS M-5, M-6
35	O'Rourke	Manifest & Cycloplegic Refraction	2 PERIODS T-5, T-6
36	Osher	Squint Surgery	2 PERIODS W-5, W-6
37	Paton	Corneal Surgery	3 PERIODS M-6, T-6, W-6
38	Payne, et al	Practical Eye Pathology	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
39	Pino	Bifocal Lenses & Presbyopia	2 PERIODS M-4, T-4
40	Romaine, et al	Ocular Motility	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
41	Rucker	Neuro-ophthalmology	4 PERIODS M-4, T-4, W-4, Th-4
42 or 43	Schepens	Ophthalmoscopy in Diagnosis of Retinal Detachment	2 PERIODS M-5, M-6 or T-5, T-6
44	Sloane, et al	Refraction	6 PERIODS T-4, T-5, W-4, W-5, Th-4, Th-5
45	Theobald, et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
46	Thorpe, et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
47	Thorpe	Surgery of Intraocular Foreign Bodies	2 PERIODS T-4, W-4
48	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-5, M-6, T-5, T-6, W-5, W-6
49	Wiener	Practical Points in Surgery	3 PERIODS M-4, T-4, W-4

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to OCTOBER 1

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

After OCTOBER 1, mail to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
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Academy Candidates and Guests May Purchase Tickets at Registration Desk Only

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 9

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

☐ Monday

☐ Tuesday

☐ Wednesday

☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1953 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the General Registration Desk in Chicago.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD	FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
	Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON. 4						
P.M. 5						
6						
TUES. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
WED. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
THURS. 1						
A.M. 2						
3						
P.M. 4						
5						
6						

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Psychosomatic Medicine
202	Alvaro	Uveitis: Etiology & Treatment
206	Baker	Neuralgias of Head
210	Berke	Surgery of Oblique Muscles
217	Chace	Ocular Malingering
218	Chamberlain	Vertical Motor Anomalies
222	Cusick	Ptosis
224	DeVoe	Corneal Dystrophies
226	Dunnington	End Results in Eye Muscle Surgery
227	Egan	Cross Cylinder Application & Theory
245	Harley	Lesions of the 3rd, 4th & 6th Cranial Nerves
250	Joy	Angiography in Ophthalmology
271	McGraw	Contact Lenses
294	Spaeth	Plastic Surgery
A	Home Study	Embryology, Anatomy & Neuro-Anatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
225	Dunlap	Divergence Anomalies
230	Fralick	Surgical Anatomy of Operations for Glaucoma
233	Fry	Lesions at Posterior Pole of Eye
238	Gipner	Recent Advances
242	Gray	Intraocular Foreign Bodies
246	Harris	Anesthetic Agents & Technics
259	Lepard	Eye Diseases Common to Children
262	Linksz	Visual Physiology
272	McGuire	Heterophoria and Heterotropia
276	Newell	Development of Refinements in Ophthalmic Surgery
296	Stimson	Lenses for the Aphakic
305	Walsh	Interesting Neuro-ophthalmologic Cases
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
205	Atkinson	Measures in Cataract Surgery
209	Berens	..... SOLD OUT .....
212	Boyes	Eye Surgery Problems
213	Burch	Psychosomatic Medicine
215	Calhoun	Dystrophies & Degenerations of Cornea
220	Chandler	Cataract Surgery: Secondary Procedures
223	Cutler	Procedures Used in Enucleation
228	Falls	Ocular Geriatrics
234	Gailey	Complications of Cataract Surgery
235	Gemeroy	External Diseases of Eye
236	Gibson	Retinopathies: Differential Diagnosis
240	Goar	Complications of Cataract Surgery
243	Haik	Beta Irradiation
247	Hogan	Pathology of the Choroid
248	Hughes	Plastic Surgery: Minor Procedures
256	Leinfelder	Papilledema & Optic Neuritis
261	Lewis	External Diseases
263	Lueck	Practical Optics
266	Mangold	Proper Fitting of Glasses
270	McCaslin	Ocular Anesthesia
274	Morrison	Office Management of Glaucoma
288	Rychener	Lacrimal Apparatus
C	Home Study	Optics & Principles of Biomicroscopy

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Psychosomatic Medicine
202	Alvaro	Uveitis: Etiology & Treatment
206	Baker	Neuralgias of Head
210	Berke	Surgery of Oblique Muscles
214	Byrnes	Adjuncts in Teaching Ophthalmology
217	Chace	Ocular Malingering
218	Chamberlain	Vertical Motor Anomalies
222	Cusick	Ptosis
224	DeVoe	Corneal Dystrophies
227	Egan	Cross Cylinder Application & Theory
229	Falls	Practical Application of Heredity
250	Joy	Angiography in Ophthalmology
271	McGraw	Contact Lenses
278	Owens & Owens	Retrolental Fibroplasia
D	Home Study	Refraction

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
225	Dunlap	Divergence Anomalies
233	Fry	Lesions at Posterior Pole of Eye
238	Gipner	Recent Advances
242	Gray	Intraocular Foreign Bodies
246	Harris	Anesthetic Agents & Technics
259	Lepard	Eye Diseases Common to Children
262	Linksz	Visual Physiology
272	McGuire	Heterophoria & Heterotropia
279	Paulson	Lighting for Comfortable & Accurate Seeing
284	Reese	Tumors & Tumor-like Lesions of the Orbit
296	Stimson	Lenses for the Aphakic
305	Walsh	Interesting Neuro-ophthalmologic Cases
E	Home Study	External Diseases, Bacteriology & Virology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
205	Atkinson	Measures in Cataract Surgery
209	Berens	Cyclodiathermy, Iridencleisis, Cyclodialysis
213	Burch	Psychosomatic Medicine
215	Calhoun	Dystrophies & Degenerations of Cornea
220	Chandler	Cataract Surgery: Secondary Procedures
223	Cutler	Procedures Used in Enucleation
234	Gailey	Complications of Cataract Surgery
235	Gemeroy	External Diseases of Eye
236	Gibson	Retinopathies: Differential Diagnosis
240	Goar	Complications of Cataract Surgery
243	Haik	Beta Irradiation
261	Lewis	External Diseases
266	Mangold	Proper Fitting of Glasses
270	McCaslin	Ocular Anesthesia
274	Morrison	Office Management of Glaucoma
283	Randolph	Handling Patients with Advanced Glaucoma
288	Rychener	Lacrimal Apparatus
289	Scheie	Emergency Eye Surgery
291	Simpson	Convergence Insufficiency & Divergence Excess
295	Steffensen	ACTH, Cortisone & Hydrocortisone
F	Home Study	Perimetry & Neuro-ophthalmology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
203	Alvis	End Results of Cataract Wound Closure
204	Amenabar	Detachment of Retina
211	Bettman	Value of Goniotomies
216	Cassady	Dacryocystitis of Infancy
237	Gifford	Dacryocystorhinostomy
241	Gordon	Surgical Importance of Orbital Fascia
254	Krewson	Paralytic Strabismus
264	Lyle	Cranial Lesions
265	Macnie	Head Trauma with Ocular Significance
269	Martens	Problems in Presbyopia
273	Meyer	Glaucoma Surgery
281	Pfeiffer	Diagnosis with Aid of X-Ray
282	Post	Operating Room Technic
298	Tabor	Vertical Muscles
301	Theodore	Ocular Therapeutics
G	Home Study	Extraocular Muscles

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
219	Chamlin	Perimeter: Types & Technics
232	Fread	Ideal Refraction Equipment
239	Givner	Systemic Disease
244	Hanser	Studies in Ocular Allergy
249	Jobe	Refraction
251	Katz	Corneal Dystrophies
257	Lemoine	Vitreous Opacities
275	Mullen	Ophthalmic Emergencies
277	Olson	Iritis, Iridocyclitis & Keratitis
280	Perera	Retinal Detachment
286	Rudolph	Advantages of General Anesthesia
292	Smith	Practical Points in Office Procedure
293	Spaeth	Lacrimal Gland & Sac
300	Tassman	Retinopathy in Diabetics
303	Trueman	Cerebral Lesions
304	Veirs	..... SOLD OUT .....
306	Wendland	Chiasmal Lesions
H	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
208	Bellows	Cataract
212	Boyes	Eye Surgery Problems
221	Cholden	Informing Patient of Blindness: Psychiatric Aspects
252	Klien	Clinical Pathology of Retinal Lesions
255	Kronfeld	Glaucoma Surgery: End Results
256	Leinfelder	Papilledema & Optic Neuritis
258	Leopold	..... SOLD OUT .....
260	Levy	Motion Picture Photography: Methods & Highlights
267	Mann, M.	Hypertensive Vascular Changes in Fundus
268	Mann, W.	Optical Correction of Aphakia
285	Robinson	Reading Retardation & Vision
287	Rundles	Streak Retinoscopy
290	Scheie	Surgery in Congenital Glaucoma
295	Steffensen	ACTH, Cortisone & Hydrocortisone
297	Swan	Ophthalmic Therapeutics
299	Tait	Accommodation & Convergence Abnormalities
307	Whalman	Minor Surgical Procedures of Eye
308	Wood	Macular Lesions
I	Home Study	Pathology

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
203	Alvis	End Results of Cataract Wound Closure
204	Amenabar	Detachment of Retina
211	Bettman	Value of Goniotomies
216	Cassady	Dacryocystitis of Infancy
237	Gifford	Dacryocystorhinostomy
241	Gordon	Surgical Importance of Orbital Fascia
253	Kornzweig	Practical Embryology
254	Krewson	Paralytic Strabismus
264	Lyle	Cranial Lesions
265	Macnie	Head Trauma with Ocular Significance
269	Martens	Problems in Presbyopia
281	Pfeiffer	Diagnosis with Aid of X-Ray
282	Post	Operating Room Technic
298	Tabor	Vertical Muscles
301	Theodore	Ocular Therapeutics
J	Home Study	Pharmacology & Therapeutics

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
207	Barkan	Congenital Glaucoma
208	Bellows	Cataract
232	Fread	Ideal Refraction Equipment
239	Givner	Systemic Disease
244	Hanser	Studies in Ocular Allergy
249	Jobe	Refraction
251	Katz	Corneal Dystrophies
257	Lemoine	Vitreous Opacities
275	Mullen	Ophthalmic Emergencies
277	Olson	Iritis, Iridocyclitis & Keratitis
280	Perera	Retinal Detachment
286	Rudolph	Advantages of General Anesthesia
292	Smith	Practical Points in Office Procedure
300	Tassman	Retinopathy in Diabetics
303	Trueman	Cerebral Lesions
304	Veirs	..... SOLD OUT .....
306	Wendland	Chiasmal Lesions

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
252	Klien	Clinical Pathology of Retinal Lesions
255	Kronfeld	Glaucoma Surgery: End Results
267	Mann, M.	Hypertensive Vascular Changes in Fundus
268	Mann, W.	Optical Correction of Aphakia
285	Robinson	Reading Retardation & Vision
287	Rundles	Streak Retinoscopy
290	Scheie	Surgery in Congenital Glaucoma
295	Steffensen	ACTH, Cortisone & Hydrocortisone
299	Tait	Accommodation & Convergence Abnormalities
307	Whalman	Minor Surgical Procedures of Eye
308	Wood	Macular Lesions

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
101	Altmann & Waltner	Histopathology of Ear—Part I	2 PERIODS W-1, W-2
102	Altmann & Waltner	Histopathology of Ear—Part II	2 PERIODS Th-1, Th-2
103 or 104	Boies, et al	Head Pain	2 PERIODS W-2, W-3 or Th-2, Th-3
105 or 106	Bryan & Bryan	Cytologic Pathology in the Differential Diagnosis of ENT Diseases	2 PERIODS T-1, T-2 or W-2, W-3
107 or 108	Clerf, et al	Diseases of the Larynx, Neck, Tracheobronchial Tree & Esophagus	2 PERIODS W-1, W-2 or Th-1, Th-2
109 or 110	Conley, et al	Cancer of Head & Neck	2 PERIODS T-1, T-2 or Th-1, Th-2
111	Converse	Nasal Plastic Surgery—Basic	2 PERIODS T-2, T-3
112	Converse	Nasal Plastic Surgery—Advanced	2 PERIODS W-2, W-3
113	Cullen, et al	Anesthesia for Otolaryngologic Surgery	2 PERIODS Th-1, Th-2
114	Fowler & Montreuil	Histology of Ear — Vertical Sections	2 PERIODS T-1, T-2
115	Fowler & Montreuil	Histology of Ear — Horizontal Sections	2 PERIODS W-1, W-2
116	Furstenberg, et al	Tumors & Infections of the Neck	2 PERIODS T-2, T-3
117 or 118	Hansel	Allergy	2 PERIODS W-2, W-3 or Th-2, Th-3
119	Hoople, et al	Fluid Ears	2 PERIODS Th-2, Th-3
120	Jackson	Peroral Endoscopy	3 PERIODS T-1, W-3, Th-1
121	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS Th-1, Th-2
122 or 123	Lindsay	Vertigo	2 PERIODS T-1, T-2 or W-1, W-2
124 or 125	Marcus, et al	Hearing Problems in Children	2 PERIODS T-1, T-2 or Th-1, Th-2
126 or 126-A	Meltzer, et al	Otological Problems	2 PERIODS T-1, T-2 or W-1, W-2
127 or 128	Moses	Speech & Voice Therapy	2 PERIODS T-1, T-2 or W-1, W-2
129	Mueller, et al	Hearing Aids	2 PERIODS W-1, W-2
130	Priest, et al	Medication for the Otolaryngologist	1 PERIOD T-1
131	Proetz, et al	Rhinological Problems	2 PERIODS W-2, W-3
132 or 133	Scheldrup	Anatomy of the Neck	2 PERIODS T-2, T-3 or W-2, W-3
134	Semenov	Therapy in Pathology of ENT	2 PERIODS Th-1, Th-2
135	Senturia, et al	External Otitis	2 PERIODS T-1, T-2
136	Tucker, et al	Esophageal Diseases & Foreign Bodies	2 PERIODS T-1, T-2
137	Tucker, et al	Laryngeal Surgery	2 PERIODS W-1, W-2

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Barone	Hay Fever
404	Bellucci	Submucous Resection of Nasal Septum
411	Carpenter & Goff	Respiratory and Swallowing Problems in the Newborn
412	Cody	The Elusive Facial Nerve
420	Day	What the Otologist Can Do for Deafness
423	Engler & Hulse	Tuberculosis of the Larynx
442	Helinger	Pediatric Laryngology
445	Hosler	Cardiac Arrest
449	Huffman	Zygomatic Fracture-Dislocations
453	Jones, L. T.	Tear Sac Surgery
461	Kinney	Acoustic Trauma
465	Lederer	Diagnostic Approaches & Recent Trends
468	Lempert	Temporal Bone Surgery
475	McQuiston	Endaural Radical Mastoidectomy
484	Pastore	A Lump in the Throat
498	Schall	Cancer of the Nose & Sinuses
499	Schuknecht	Carcinoma of the Oral Cavity & Jaws
504	Simmons	Intranasal Dacryocystorhinostomy
505	Simonton	Recruitment of Loudness and Diplacusis Binauralis
509	Sullivan	Facial Paralysis and Bell's Palsy
511	Tingwald	Tumors of the Paranasal Sinuses
514	Van Alyea	Chronic Sinusitis

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
415	Corgill	Laryngopharyngectomy & Bilateral Dissection
428	Glick	The Sinuses
432	Hallberg	Severe Nosebleed
435	Harris	Poliomyelitis: Respiratory Complications
439	Hilger	Therapeutic Approaches to the Inner Ear
447	House	Atresia of the Ear Canal
450	Ireland	Common Sense Treatment of Office Patients
454	Jordan, R. E.	Allergy in Otology
460	Kemler	Mirror Laryngoscopy
466	LeJeune	Intralaryngeal Surgery
472	McGannon	Respiratory Allergy
476	Miller	Laryngeal Emergencies in Infants & Children
480	Nash	Otolaryngologic Mishaps
485	Pearlman	ENT Complaints in Old Age
488	Putney	Surgery of Neck Tumors
490	Reger	Detection of Auditory Malingering
496	Rosenberger	Practical Aspects of Childhood Otology
500	Sellers	Headache
515	Walsh	Acute & Chronic Otitis Media
517	Work	Wide Field Laryngectomy

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
401	Ashley	Dust & Other Perennial Inhalant Therapy
405	Bordley	Hearing Disorders in Preschool Children
407	Brandenburg	Understanding the Problem Patient
410	Carhart	Audiogram in Otologic Diagnosis
414	Cohen	Rhinoplasty Complications
419	Davison	Bronchology & Bacterial Allergy
421	Derlacki	Role of Allergy in Deafness
427	Forman	Problems in Laryngoscopy
430	Glorig	Construction of a Practical Soundproof Room
433	Hampsey	Respiratory Allergy
434	Harrill	Bronchography
438	Hilger	Direct Fixation of Facial Fractures
440	Hill	Antro-Alveolar Fistulas
443	Holinger	Esophageal Diseases in Infants & Children
444	Holmes	Scars of the Face
451	Johnson	Anesthesia in Adult Tonsillectomies
456	Juers	Hearing Tests
463	Lake	Chronic Suppurative Otitis Media
467	Lempert	Diagnosing Type of Hearing Loss
470	Macgregor	Psychological Hazards of Plastic Surgery of the Face
471	Maxwell	Tumors of the Face & Neck in Infancy & Childhood
477	Missal	Skin Tests in Allergy
478	Moore	Eustachian Tube Problems
486	Perlman	Serous Otitis
487	Proetz	Clinical Aspects of Nasal Respiration
492	Richards	Suggestions Concerning Office Practice
501	Seltzer	Plastic Surgery
502	Shambaugh	Practical Office Routine in Otologic Problems
510	Thornell	Intralaryngeal Arytenoidectomy
516	Weille	Nasal & Sinus Surgery in Respiratory Allergy

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Barone	Hay Fever
403	Becker	Plastic Surgery of Protruding Ears
404	Bellucci	Submucous Resection of Nasal Septum
411	Carpenter & Goff	Respiratory and Swallowing Problems in the Newborn
412	Cody	The Elusive Facial Nerve
416	Costen	Mandibular Joint Syndrome
418	Crispell	Allergy in Children
419	Davison	Bronchology & Bacterial Allergy
420	Day	What the Otologist Can Do for Deafness
423	Engler & Hulse	Tuberculosis of the Larynx
431	Goodhill	Management of Tinnitus
437	Hickey	Functional or Organic Nervous Disease
440	Hill	Antro-Alveolar Fistulas
442	Holinger	Pediatric Laryngology
449	Huffman	Zygomatic Fracture-Dislocations
453	Jones, L. T.	Tear Sac Surgery
454	Jordan, R. E.	Allergy in Otology
474	McNally	Labyrinthine Tests
479	Morrison	Laryngeal Paralysis
489	Rawlins	ACTH, Cortisone & the Antihistaminic Drugs
495	Robison	Otitis Media with Effusion
498	Schall	Cancer of the Nose & Sinuses
499	Schuknecht	Carcinoma of the Oral Cavity & Jaws
503	Shuster	Vestibular Apparatus: Clinical Considerations
508	Slaughter	Blood Vessel Tumors of Head & Neck
512	Tremble	Practical Therapeutics in Office Practice

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
406	Boyden	Chronic Frontal Sinus Surgery
415	Corgill	Laryngopharyngectomy & Bilateral Dissection
425	Fabricant	Modern Medication of Nose & Sinuses
426	Figi	Tumors of Ear & Mastoid
432	Hallberg	Severe Nosebleed
436	Hedgecock	Advanced Clinical Audiometry
448	Howard	Office Procedures in ENT
450	Ireland	Common Sense Treatment of Office Patients
455	Jordan, V.M.	Sudden Death in the Operating Room
458	Kelemen	Tonsillar Tissue
462	Kos	Clinical Orientation in Management of Dizziness
467	Lempert	Diagnosing Type of Hearing Loss
482	Neivert	Workshop for Deformed Instruments
493	Rinkel	Food Allergy in Otolaryngologic Practice
497	Ryan	Headache
500	Sellers	Headache
505	Simonton	Recruitment of Loudness & Diplacusis Binauralis
506	Singleton	Acute Otitis Media
515	Walsh	Acute & Chronic Otitis Media
518	Wynn	Facial Injuries: Plastic Repair

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
401	Ashley	Dust and Other Perennial Inhalant Therapy
405	Bordley	Hearing Disorders in Preschool Children
407	Brandenburg	Understanding the Problem Patient
408	Brodnitz	The Actor & Singer in ENT Practice
409	Canfield	Audiology for Practicing Otologist
417	Craft	Dermatologic Allergy
422	DeWeese	The Dizzy Patient
424	Erich	Fractures of Facial Bones
427	Forman	Problems in Laryngoscopy
433	Hampsey	Respiratory Allergy
441	Hilsinger	Vascular Headaches
444	Holmes	Scars of the Face
446	House	Surgery of Chronically Discharging Ear
452	Jones, A. C.	Helpful Hints for Everyday Practice
457	Keaster	Speech & Hearing in Children Under Five
463	Lake	Chronic Suppurative Otitis Media
464	Lassman	Prescription of Hearing Aids
469	Levin	Management of Defects of Speech & Voice
470	Macgregor	Psychological Hazards of Plastic Surgery of the Face
473	McLaurin	Otitis Externa: Therapy
483	Ogura	Laryngectomy & Neck Dissection
492	Richards	Suggestions Concerning Office Practice
494	Roberts	Medical Otolaryngology
501	Seltzer	Plastic Surgery
509	Sullivan	Facial Paralysis and Bell's Palsy
510	Thornell	Intralaryngeal Arytenoidectomy

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
403	Becker	Plastic Surgery of Protruding Ears
413	Cohen	Deviated Noses & Their Correction
416	Costen	Mandibular Joint Syndrome
418	Crispell	Allergy in Children
428	Glick	The Sinuses
431	Goodhill	Management of Tinnitus
437	Hickey	Functional or Organic Nervous Disease
439	Hilger	Therapeutic Approaches to the Inner Ear
443	Holinger	Esophageal Diseases in Infants & Children
452	Jones, A. C.	Helpful Hints for Everyday Practice
460	Kemler	Mirror Laryngoscopy
466	LeJeune	Intralaryngeal Surgery
471	Maxwell	Tumors of the Face & Neck in Infancy & Childhood
472	McGannon	Respiratory Allergy
474	McNally	Labyrinthine Tests
475	McQuiston	Endaural Radical Mastoidectomy
478	Moore	Eustachian Tube Problems
483	Ogura	Laryngectomy & Neck Dissection
484	Pastore	A Lump in the Throat
489	Rawlins	ACTH, Cortisone & the Antihistaminic Drugs
493	Rinkel	Food Allergy in Otolaryngologic Practice
503	Shuster	Vestibular Apparatus: Clinical Considerations
504	Simmons	Intranasal Dacryocystorhinostomy
507	Skolnik	Cleft Palate
508	Slaughter	Blood Vessel Tumors of Head & Neck
513	Van Alyea	Embryology of ENT

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
425	Fabricant	Modern Medication of Nose & Sinuses
426	Figi	Tumors of Ear & Mastoid
435	Harris	Poliomyelitis: Respiratory Complications
445	Hosler	Cardiac Arrest
447	House	Atresia of the Ear Canal
455	Jordan, V.M.	Sudden Death in the Operating Room
459	Kelemen	Mechanical Injuries of Hearing Organ
462	Kos	Clinical Orientation in Management of Dizziness
465	Lederer	Diagnostic Approaches & Recent Trends
476	Miller	Laryngeal Emergencies in Infants & Children
482	Neivert	Workshop for Deformed Instruments
485	Pearlman	ENT Complaints in Old Age
487	Proetz	Clinical Aspects of Nasal Respiration
491	Reger	Practical Office Audiometry
495	Robison	Otitis Media with Effusion
496	Rosenberger	Practical Aspects of Childhood Otology
502	Shambaugh	Practical Office Routine in Otologic Problems
506	Singleton	Acute Otitis Media
511	Tingwald	Tumors of the Paranasal Sinuses
512	Tremble	Practical Therapeutics in Office Practice
517	Work	Wide Field Laryngectomy

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
408	Brodnitz	The Actor & Singer in ENT Practice
409	Canfield	Audiology for Practicing Otologist
410	Carhart	Audiogram in Otologic Diagnosis
417	Craft	Dermatologic Allergy
421	Derlacki	Role of Allergy in Deafness
422	DeWeese	The Dizzy Patient
424	Erich	Fractures of Facial Bones
429	Glorig	Terms Used in Audiology
434	Harrill	Bronchography
436	Hedgecock	Advanced Clinical Audiometry
441	Hilsinger	Vascular Headaches
446	House	Surgery of Chronically Discharging Ear
448	Howard	Office Procedures in ENT
451	Johnson	Anesthesia in Adult Tonsillectomies
456	Juers	Hearing Tests
461	Kinney	Acoustic Trauma
464	Lassman	Prescription of Hearing Aids
468	Lempert	Temporal Bone Surgery
473	McLaurin	Otitis Externa: Therapy
477	Missal	Skin Tests in Allergy
479	Morrison	Laryngeal Paralysis
481	Nash	Functional Diseases of Nose
486	Perlman	Serous Otitis
488	Putney	Surgery of Neck Tumors
494	Roberts	Medical Otolaryngology
497	Ryan	Headache
518	Wynn	Facial Injuries: Plastic Repair

News - - - Notes

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

The regular Spring Meeting of the New England Oto-Laryngological Society was held at the Massachusetts Eye and Ear Infirmary in Boston on June 17, Dr. LeRoy A. Schall and Dr. John A. Murtagh presiding. Among the speakers were Drs. Alan S. Feldman, Jarrett H. Folley, Otis F. Jillson and Eugene G. Boss. Dr. Andrew A. Eggston, the guest speaker, discussed "Malignancies of the Ear."

The next meeting of the society will be held on Wednesday, November 18, 1953.

CALIFORNIA

The Research Study Club of Los Angeles announces its Twenty-Third Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 18 through 29, 1954. The guest lecturers for otolaryngology will be Dr. Lawrence R. Boies and Dr. Jerome A. Hilger of the University of Minnesota, Dr. Paul H. Holinger of the University of Illinois, and Dr. Lewis F. Morrison of the University of California. In ophthalmology the guest lecturers will include Dr. Daniel B. Kirby, Professor of Ophthalmology, New York University, and Dr. Frank D. Costenbader of Washington, D. C.

The first week, January 18 through 23, will be devoted to otolaryngology. The ophthalmology section will begin Monday, January 25, and end on Friday, January 29.

The fee for the Clinical Course is $100 and should be sent to Dr. Pierre Viole, Treasurer, 1930 Wilshire Boulevard, Los Angeles 5, California.

COLORADO

The summer convention of the Colorado Ophthalmological Society will be held on August 3 to 6, 1953, in conjunction with the Department of Graduate Education of the University of Colorado Medical School. Guest speakers, seminars, and demonstrations will constitute the four-day meeting, to be held at the University of Colorado Medical Center in Denver. Membership in the Society and attendance at the meeting is open to all interested and accredited physicians. For information on registration and fees, write to the Director of Graduate Education, University of Colorado Medical School, 4200 E. Ninth Avenue, Denver, Colorado.

ILLINOIS

The Chicago Ophthalmological Society held its regular meeting on May 18, 1953. Dr. Arlington C. Krause presented a paper on "Toxoplasmosis" during the instruction hour. On the scientific program were Drs. Beulah Cushman, Robert L. Willard, R. Tully Bradford, and Lawrence J. Lawson, Jr.

MARYLAND

On June 9, 1953 Dr. Frederick H. Verhoeff was given the Doctor of Laws degree by Johns Hopkins University. The accompanying remarks are those of Dr. Alan C. Woods, Chief of Ophthalmology at the Johns Hopkins Hospital.

Mr. President:

On behalf of the Faculties of the University and with the approval of the Trustees, I have the honor to present a candidate for the degree of Doctor of Laws.

Dr. Verhoeff was a graduate of Yale University in 1895, and of the Johns Hopkins University School of Medicine in 1899. He is the first graduate of this school to become an ophthalmologist. While still an undergraduate, he began his researches on binocular vision, which have been continued throughout his life and have contributed much to our knowledge in this field. While he has achieved great distinction as a clinician, his major interest has been in research in the realm of ophthalmic pathology. He may well be termed the American pioneer in this field. He became Resident Pathologist at the Massachusetts Eye and Ear Infirmary in 1900 and later Pathologist to this Institution. Appointed to the Staff of Harvard University in 1900, he advanced through various academic grades and became Professor of Ophthalmic Research in 1924. He is now Professor Emeritus.

His researches in ophthalmology and contributions to our knowledge have been many. He has established the actual pathogenesis of previously mysterious ocular inflammations. His investigations on the histologic and metabolic changes produced by a great variety of ocular diseases, and on the physiology and chemistry of the eye have yielded results which may well be called classic and are now part of our established knowledge. He has been honored by ophthalmological societies throughout this nation, holding the highest offices and receiving the most distinguished awards they can bestow. His entire life has been unselfishly devoted to the advancement of his science, and to mankind's war against blindness. He has reflected honor on the University and the Institutions where he has held office, and on his own Alma Mater, the Johns Hopkins University, where he received his medical training.

It is my pleasure and honor to nominate to you Dr. Frederick Herman Verhoeff as a candidate for the degree of Doctor of Laws.

MINNESOTA

The Minnesota Academy of Ophthalmology and Otolaryngology met on May 8, 1953, in Minneapolis. On the program were Dr. Anderson C. Hilding, Duluth, whose subject was "The Postoperative Appearance of the Hyaloid Membrane as Seen on Slitlamp Examination and Its Possible Relation to Fibroblastic Invasion," and Dr. Robert J. Priest and Dr. W. J. Kucera, Jr., Minneapolis, who described "The Air Sac of the Ape Occurring in Man."

MISSOURI

The Annual Spring Meeting of the Kansas City Society of O.O.R.L. was held in Springfield, Missouri, on May 16, 1953. Dr. Arthur J. Bedell of Albany, New York, was the guest speaker and presented a paper entitled "Do Not Use Atropin in Traumatic Hyphaema."

Dr. Bernard Becker, at present at the Wilmer Institute, has been appointed Professor and Head of the Department of Ophthalmology, Washington University, replacing Dr. Lawrence T. Post, who retired from these duties July 1, 1953. Since Dr. Becker will be unable to assume his duties until about January 1, 1954, Dr. T. E. Sanders has been appointed Acting Head of the Department.

PENNSYLVANIA

Dr. William M. Hart, Assistant Professor of Ophthalmology of Jefferson Medical College, Philadelphia, has been appointed Chief of the Ophthalmology Clinics of the National Institute of Neurological Diseases and Blindness, Public Health Service. His appointment began as of April 15.

The VI Annual Clinical Conference of the Wills Eye Hospital will be held on February 19 and 20, 1954. Color television sponsored by Smith, Kline and French will again be a feature of the program.

VIRGINIA

The Gill Memorial Eye, Ear and Throat Hospital, Roanoke, Va., has just completed the Twenty-Sixth Annual Refresher Course in ophthalmology and otolaryngology. There were about three hundred doctors in attendance. Forty-five states were represented including Canada and several foreign countries.

The Twenty-Seventh Annual Spring Congress will be held from April 5 to 10, 1954.

BRAZIL

Manoel A. da Silva Foundation

On May 15, 1953 a photograph of Dr. Manoel A. da Silva was hung in the room which bears his name, that is, in the library of the Ophthalmological Study Center (Sao Paulo, Brazil).

On this occasion Dr. Jair Xavier Guimaraes in the name of Manoel A. da Silva's medical school classmates reminded those present of Dr. Silva's record up to the time of his death in 1950, at the age of 36. Dr. Renato de Toledo, Chief of Staff of the Department of Ophthalmology of the Escola Paulista de Medicina, also said a few words about the deceased. Prof. Moacyr E. Alvaro, Director of the Department of Ophthalmology of the Escola Paulista de Medicina, referred to the generous gesture of Dr. Manoel A. da Silva's parents, who have willed their entire estate for the establishment of the Manoel A. da Silva Foundation, which is dedicated to the advancement of the teaching of ophthalmology. The Manoel A. da Silva Foundation will carry out its work through the Ophthalmological Study Center and the Department of Ophthalmology of the Escola Paulista de Medicina.

POSITIONS AVAILABLE

There are vacancies available for a Junior and a Senior Resident in Otolaryngology at the Veterans Administration Center, Wood, Wisconsin. The residency is approved for three years and is supervised by the consultants, who are representatives of the Dean's Committee of Marquette University Medical School. A Basic Science course may be included in the residency program at the expense of the Veterans Administration. Salary ranges from $2700 to $3300 per annum. Veteran physicians are preferred but not essential.

For further information write to Dr. R. H. Lehman, Chief, Departments of Ophthalmology and Otolaryngology, Veterans Administration Center, Wood, Wisconsin.

A fellowship has been created by the Retina Foundation and will be granted to a young ophthalmologist from the United States, or abroad, who wishes to be trained for six to twelve months at the Retina Service of the Massachusetts Eye and Ear Infirmary. The remuneration will amount to $1000 per annum, plus room, board, and laundry at the Infirmary.

Training covers methods of examination of the extreme fundus periphery and the vitreous; use of the indirect stereoscopic ophthalmoscope and scleral depressor; and surgical procedures for cases of retinal detachment. Original research work of clinical nature will be encouraged. The only candidates who are eligible are those who have completed a residency in ophthalmology or have had comparable training.

Applications accompanied with a biography and at least two references should be mailed to Dr. E. B. Dunphy, Chief of Ophthalmology, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston 14, Mass.

Resident, otorhinolaryngology: 500 bed hospital, medical school affiliation. Maintenance and $100.00 monthly. Vacancy in September. For information write Otto C. Hirst, M.D., Duval Manor, Johnson and Greene Sts., Philadelphia 44, Pa.

Needed, a specialist in both eye and ear, nose and throat in established clinic, with early prospect of partnership status. Equipment furnished. For further information write Dr. Louis F. Pine, Lake Region Clinic, Devils Lake, North Dakota.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

International Association of Eye, Ear, Nose and Throat Society Secretaries

President: Dr. James J. Monahan
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.
Time and Place: 5:30 p.m., October 13, 1953. Palmer House, Chicago

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Prof. A. Franceschetti, rue A—Jentzer 22, Geneva, Switzerland

International Broncho-Esophagological Society

President: Dr. Fernand Eeman
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut Street, Philadelphia 3, Pa.

International Council of Ophthalmology

President: Sir Stewart Duke-Elder
Secretary: Prof. Dr. Edward Hartmann, 2, Avenue Ingres, Paris (16e) France
Time and Place: The Council meets at the time and place of each International Congress (usually every 4 years) and other years alternately in France and Great Britain

International Organization Against Trachoma

President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

Pan-American Association of Ophthalmology

President: Dr. M. E. Alvaro
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: February 1956. Santiago, Chile

Pan-American Association of Ophthalmology, Puerto Rico Chapter

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut Street, Philadelphia 3, Pa.
Time and Place: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology. February 28 to March 4, 1954, Mexico City

FOREIGN

The Ophthalmological and Otolaryngological Society of Alberta

President: Dr. D. S. Gorrell
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: Spring of 1954 in Calgary, Alberta, Canada

All-India Ophthalmological Society

President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

Associacao Medica do Instituto Penido Burnier

President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Alberto Galo, 1st
Second Secretary: Dr. Aloisio Af. Ferreira

Austrian Otolaryngological Society, Vienna

President: Prof. Dr. E. Schlander
Secretary: Dr. Julius Popper, Wien I., Schottenring 17, Austria
Time and Place: First Tuesday of the month. Vienna General Hospital, Vienna IX., Alserstrasse 4

Bombay Ophthalmologists' Association

Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

British Association of Otolaryngologists

President: Mr. V. E. Negus
Honorary Secretary: Mr. Myles L. Formby, 45 Lincoln's Inn Fields, London W. C. 2, England

British Medical Association, Section on Ophthalmology

President: Mr. J. H. Doggart
Secretary: Mr. B. Gluck, 76 Cathedral Road, Cardiff
Time and Place: July 17, 1953. Cardiff

Canadian Medical Association, Section on Ophthalmology

Secretary: Dr. J. Clement McCulloch, 830 Medical Arts Building, Toronto, Ont.

Canadian Ophthalmological Society

President: Dr. Kenneth B. Johnston
Secretary: Dr. R. G. C. Kelly, 113 St. Clair Avenue W., Toronto 5, Ont., Canada

Canadian Otolaryngological Society

President: Dr. D. E. S. Wishart
Secretary: Dr. W. Ross Wright, 361 Regent Street, Fredericton, New Brunswick

Centro de Estudos de Oftalmologia de Sao Paulo

President: Dr. Rubens Belfort Mattos
Secretary: Dr. Jose Carlos Gouvea Pacheco, Marconi 34, Sao Paulo

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

CHENGTU OPHTHALMOLOGICAL SOCIETY

President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

CHINESE OPHTHALMOLOGY SOCIETY

President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEKING

President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG

President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

FACULTY OF OPHTHALMOLOGISTS

President: Mr. J. H. Doggart
Secretary: Mr. E. F. King, 45 Lincoln's Inn Fields, London, W.C. 2, England

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY

President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY

President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

ISRAEL OPHTHALMOLOGICAL SOCIETY

President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. N. P. R. Galloway
Secretary: Dr. R. D. Weeden Butler, c/o Birmingham & Midland Eye Hospital, Church St., Birmingham 3, England
Place: Birmingham & Midland Eye Hospital, Church St., Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP

President: Prof. Dr. H. M. Dekking
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, Gravenhage, The Netherlands

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

NORSKE OYELEGERS FORENING (SOCIETY OF NORWEGIAN OPHTHALMOLOGISTS)

President: Dr. J. C. Holst, Pilestredet 28, Oslo
Secretary: Dr. Thore Lie Thomassen, Rikshospitalets Oeyenklinikk, Oslo

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. W. Kirkpatrick
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. L. Tostevin
Secretary: Dr. Arnold L. Lance, 27 Commonwealth Street, Sydney, New South Wales, Australia
Time and Place: October 12, 1953, Adelaide, South Australia

DET OFTALMOLOGISKE SELSKAB I KOBENHAVN (DENMARK)

President: Dr. G. Osterberg
Secretary: Dr. S. Ry Andersen, Vestre Boulevard 41, Copenhagen, Denmark
Place: Rigshospitalets ojenafdeling, Copenhagen, Denmark

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Dr. Ahmad Kamel
Honorary Secretary: Dr. Sabri Kamel, Dar El Hekmah, 42 Ksar El Ainy Street, Cairo, Egypt
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ

Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Nuestra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

President: Dr. Howard Coverdale
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

President: Dr. L. Staz
Secretary-Treasurer: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderley Street, Cape Town
Time: Annually

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Dr. A. MacRae
Honorary Secretaries: Mr. A. G. Leigh and Mr. Arthur Lister, 45 Lincoln's Inn Fields, London W. C. 2

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN

President: Prof. K. Lindner
Secretary: Dr. W. Funder, Wien IX., Alserstrasse 4, II. Univ. Augenklinik
Time and Place: Jeden 2. Montag im Monat, 19°° Wien IX., Alserstrasse 4, im Hörsaal der II. Augenklinik.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Mr. Frank W. Law
Honorary Secretary and Treasurer: Mr. Ian C. Fraser, 21 Dogpole, Shrewsbury, Salop.
Time and Place: July 6, 7 and 8, 1953. Sir William Dunn School of Pathology, Oxford

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Jesus Eusebio
Secretary-Treasurer: Dr. Jesus Tamesis, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY

Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H E Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. J. A. Conway
Secretary: Dr. John D. Fraser, 2 Bower St., Glasgow, W. 2, Scotland
Time and Place: Last Saturday of March, Edinburgh and last Saturday in October, Glasgow

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

President: Dr. Jorge Malbran
Secretary: Dr. Jose Maria Roveda, Lezica 4472, Buenos Aires
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA

President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORHINOLARINGOLOGIA

President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Elpidio Posada V., Calle 22, No. 4-75, Bogota, Colombia
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA

President: Dr. Gilberto Cepero Garcia
Secretary: Dr. Jose Alfonso, Jr.
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA

President: Dr. Manuel de Rivas Cherif
Secretary: Dr. Emilio Remolina Lopaz, Apartado Postal No. 8112, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD NICARAGUENSE DE OFTALMOLOGIA

President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA

Chairman: Dante J. Yadarola
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA

President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jalisco, Mexico
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO-AMERICANA

President: Prof. Mariano Soria Escudero
Secretary: Dr. Carlos Costi Garcia de Tunon Montalban, 3, Madrid, Spain

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL

President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO

President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzun O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA

President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA

President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladeira Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Enrico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAES

President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Est. Minas Geraes

SOCIEDADE DE OFTALMOLOGIA DEL NORTE

President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA
President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RINO-LARINGOLOGIA DO RIO GRANDE DO SUL
President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Su'
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO
President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevedo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO
President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA
President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE
President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time and Place: Last Sunday of February, June and November, Bruxelles

SOCIETE FRANCAISE D'OPHTHALMOLOGIE
Secretaire-général: Dr. Marcel Kalt, 81 rue Saint Lazare, Paris (9) France
Time and Place: May 23 to 27, 1954. Paris

SOCIETE FRANCAISE D'OTO-RHINO-LARYNGOLOGIE
Secretary-General: Dr. H. Flurin, 19 rue MacMahon, Paris, France
Time and Place: October 26-30, 1953. Paris, France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS
President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm So, Sweden
Time: Annual meeting, beginning of June

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND
President: Dr. E. C. Zorab
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, Hants, England

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Frederick C. Cordes
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 11-16, 1953, Chicago

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION
President: Dr. Edwin N. Broyles
Secretary: Dr. F. Johnson Putney, 255 South 17th Street, Philadelphia 3, Pa.
Time and Place: Annual meeting, May 25 and 26, 1954. Statler Hotel, Boston, Mass.

AMERICAN LARYNGOLOGICAL ASSOCIATION
President: Dr. Gordon F. Harkness
Secretary: Dr. Harry P. Schenck, 326 S. 19th Street, Philadelphia 3, Pa.
Time and Place: May 20 and 21, 1954, Hotel Statler, Boston, Mass.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.
President: Dr. LeRoy A. Schall
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.
Time and Place: May 25 to 27, 1954, Boston, Mass.

EASTERN SECTION
Vice-President: Dr. Irl H. Blaisdell
Secretary: Dr. F. Johnson Putney, 2029 Delancey St., Philadelphia 3, Pa.

MIDDLE SECTION
Vice-President: Dr. French K. Hansel
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill

SOUTHERN SECTION:
Vice-President: Dr. W. W. Wilkerson, Jr.
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.

WESTERN SECTION:
Vice-President: Dr. Alden H. Miller
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Trygve Gundersen
Secretary: Dr. Harold G. Scheie, 313 S. 17th St., Philadelphia 3, Pa.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY
Chairman: Dr. Dean M. Lierle
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tenn.

AMERICAN OPHTHALMOLOGICAL SOCIETY
President: Dr. William L. Benedict
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.
Time and Place: Annual meeting, June 16, 17 and 18, 1954. Many Glacier Hotel, Glacier Park, Mont.

AMERICAN OTOLOGICAL SOCIETY
President: Dr. Frederick T. Hill
Secretary-Treasurer: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.

AMERICAN OTORHINOLOGIC PLASTIC SURGERY SOCIETY
President: Dr. Harry Neivert
Secretary: Dr. Louis Joel Feit, 66 Park Avenue, New York 16, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY
President: Dr. Kenneth L. Craft
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo, N. Y.
Time and Place: Annual meeting, October 16, 1953, Chicago

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.
Chairman: Dr. Brittain F. Payne
Secretary-Treasurer: Dr. Lorand V. Johnson, 10515 Carnegie Ave., Cleveland 6, Ohio

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.
Time and Place: March 10 to 12, 1954, St. Louis, Mo.

SOCIETY OF MILITARY OPHTHALMOLOGISTS
President: Col. Austin Lowrey
Secretary-Treasurer: Col. J. H. King, Jr., Walter Reed Army Hospital, Washington 12, D. C.
Time: October, 1953.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. Joel A. Wilkinson
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, September through June. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Tadao Hata, 1704 South King Street, Honolulu, Hawaii
Secretary: Dr. Albert Ho
Time and Place: Third Thursday of each month, October through June (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Robert G. Snow
Secretary-Treasurer: Dr. Whitney J. Haight, 1015 Boston Building, Salt Lake City 1, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Ralph H. Riggs
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY
President: Dr. Wendell L. Hughes
Secretary: Dr. Ward L. Mould, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Fourth Monday in February, April, September and November at 7:30 p.m. at Meadowbrook Hospital, Hempstead, N. Y.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Merrill J. Kim
Secretary: Dr. Karl Riemer, 403 Commonwealth Avenue, Boston, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. John A. Murtagh
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Fall meeting, November 18, 1953, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Mass.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Leland G. Hunnicutt
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave. N.E., Seattle 5, Wash.
Time and Place: 1954 at Honolulu

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. F. J. Cady
Secretary: Dr. V. E. Cortopassi, 324 S. Washington Ave., Saginaw, Mich.
Time and Place: 7:00 p.m. every second Friday of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY MEDICAL SOCIETY
President: Dr. W. Howard Morrison
Secretary-Treasurer: Dr. W. C. Boden, 909 Badgerow Building, Sioux City, Iowa
Time and Place: February, 1954, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. K. W. Cosgrove
Secretary: Dr. F. A. Holden, Medical Arts Building, Baltimore, Md.
Time and Place: November, 1954, St. Louis Convention

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Louis Leo
Secretary: Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Va.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. H. J. Belson
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Ralph Clements
Secretary-Treasurer: Dr. David McCoy, 51 Woodward Building, Birmingham, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Robert C. McNaught
Secretary: Dr. Alfred R. Robbins, 1930 Wilshire Blvd., Los Angeles, Calif.
Place: 1930 Wilshire Blvd., Los Angeles, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. Fritz Nelson
Secretary: Dr. James C. Strong, Jr., 550 Metropolitan Building, Denver, Colo.
Time and Place: First Saturday of each month, October through May, University of Colorado School of Medicine, Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY
President; Dr. John Bradley
Secretary: Dr. Robert C. Shattuck, 406 Republic Building, Denver, Colo.
Time and Place: First Saturday of each month, October through May, University of Colorado School of Medicine, Denver

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Mozart A. Lischkoff
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 South Kuhl Avenue, Orlando, Fla.
Time and Place: Midwinter meeting—January, Miami Beach, Fla. Annual meeting—April, Hollywood, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. Eugene Matthews
Secretary: Dr. Alton V. Hallum, 245 Doctors Building, Atlanta 3, Ga.
Time and Place: First Friday and Saturday of March. General Oglethorpe Hotel, Savannah

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Myron S. Harding
Secretary-Treasurer: Dr. John R. Swan, 23 East Ohio Street, Indianapolis 4, Ind.
Time: Annually in May

IOWA ACADEMY OF OTOLOGY AND OTOLARYNGOLOGY
President: Dr. Oral L. Thorburn
Secretary: Dr. Jack V. Treynor, 532 First Avenue, Council Bluffs, Iowa
Time and Place: September, 1953. Ames, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George F. Gsell
Secretary: Dr. David P. Trimble, 2nd Floor, Gazette Bldg., Emporia, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. John E. McKeigue, 403 Commonwealth Avenue, Boston 15, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Archie Olson
Secretary-Treasurer: Dr. R. W Holmen, Lowry Medical Arts Building, St. Paul 2, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. Charles LaPierre
Secretary-Treasurer: Dr. H. L. Casebeer, Casebeer Hospital and Clinic, Butte, Mont.
Place: Butte, Mont.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Maynard A. Wood
Secretary-Treasurer: Dr. Paul L. Peterson, 702 Sharp Building, Lincoln, Neb.
Time and Place: May, 1954, Lincoln and 1955, Omaha

NEW JERSEY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Henry B. Orton
Secretary: Dr. Albert F. Moriconi, 438 Hamilton Avenue, Trenton 9, N. J.

MEDICAL SOCIETY OF NEW JERSEY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Robison D. Harley
Secretary: Dr. R. F. Roh, 198 Clinton Avenue, Newark, N. J.

NEW JERSEY OPHTHALMOLOGICAL SOCIETY
President: Dr. Robison D. Harley
Secretary: Dr. Charles E. Jaeckle, 136 Evergreen Place, East Orange, N. J.

NEW MEXICO OPHTHALMOLOGICAL SOCIETY
President: Dr. Howard B. Peck
Secretary-Treasurer: Dr. J. A. Dillahunt, 106 Girard S.E., Albuquerque, N. M.
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. MacLean B. Leath
Secretary-Treasurer: Dr. George B. Ferguson, 1110 West Main St., Durham, N. C.
Time and Place: September 14 to 16, 1953, Francis Marion Hotel, Charleston, S. C. (Jointly with South Carolina Society of Ophthalmology and Otolaryngology)

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Harold U'Ren
Secretary: Dr. George E. Chamberlain, 909 Medical Dental Building, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May, Columbia Athletic Club, Portland, Ore.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Samuel T. Buckman
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. H. Font
Secretary: Dr. Luis A. Morales, Calle Concordia 40, Ponce, Puerto Rico
Time and Place: Monthly, except July and August, Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Clay W. Evatt
Secretary-Treasurer: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: September 14 to 16, 1953, Francis Marion Hotel, Charleston, S. C. (Jointly with North Carolina Eye, Ear, Nose and Throat Society)

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Roland H. Myers
Secretary-Treasurer: Dr. John T. Bryan, 2105 Hayes Street, Nashville, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Dan Brannin
Secretary: Dr. Lyle Hooker, 1013 Esperson Building, Houston, Texas
Time and Place: December 4 and 5, 1953, Fort Worth, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. D. Morgan
Secretary-Treasurer: Dr. G. Slaughter Fitz-Hugh, 104 East Market St., Charlottesville, Va.

WASHINGTON STATE MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George A. Hanson
Secretary-Treasurer: Dr. Claude K. Miller, 601 Douglas St., Wenatchee, Wash.
Time and Place: September 12 to 16, 1953, Seattle, Wash.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James K. Stewart
Secretary-Treasurer: Dr. Frederick C. Reel, 404 Medical Arts Building, Charleston, W. Va.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Gerald Fonda
Secretary: Dr. Francis J. Grant, 930 Salem Avenue, Hillside, N. J.
Time and Place: 8:45 p.m., second Monday of the month, October to May. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Thayer Parry
Secretary-Treasurer: Dr. D. L. Gordon, 328 Ohio Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January, March, May and November. Akron City Club

ALTA CALIFORNIA OPHTHALMOLOGIC SOCIETY
President: Dr. Claude Gray
Secretary: Dr. Alfons F. Tipshus, 242 N. Sutter, Stockton, Calif.
Time and Place: Last Thursday of month. Sutter Club, Sacramento, Calif.

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY
President: Dr. Webb Chamberlain
Secretary-Treasurer: Dr. Joseph Krug, 988 Fifth Avenue, New York, N. Y.
Time and Place: Annual Spring Meeting each year in April, New York

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. James T. King
Secretary-Treasurer: Dr. Nathan I. Gershon, 727 W. Peachtree St. N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION
Chairman: Dr. Harold C. Dix
Secretary-Treasurer: Dr. Frederic M. Reese, 330 North Charles Street, Baltimore 1, Md.
Time and Place: 8:00 p.m., fourth Thursday of every month October to May. Medical and Chirurgical Faculty, 1211 Cathedral St.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, 501 Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through April. THE CLUB—Top of Red Mt., Birmingham

Boston City Hospital Aural and Ophthalmic Association

President: Dr. B. Riseman
Secretary: Dr. Arthur Gorney, 14 Charlesgate West, Boston, Mass.
Time and Place: Annual meeting in March, Boston City Hospital

Brooklyn Ophthalmological Society

President: Dr. Daniel K. Kravitz
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

Buffalo Ophthalmologic Club

President: Dr. Meyer H. Riwchun
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

Central Illinois Society of Ophthalmology and Otolaryngology

President: Dr. Philip R. McGrath
Secretary-Treasurer: Dr. Alfred G. Schultz, 237 East State Street, Jacksonville, Ill.
Time and Place: September 11, 1953, Abraham Lincoln Hotel, Springfield, Ill.

Central New York Eye, Ear, Nose and Throat Society

President: Dr. Alfred W. Doust
Secretary-Treasurer: Dr. Joseph Frank, University Building, Syracuse, N. Y.
Time and Place: Quarterly, University Club, Syracuse, N. Y.

Chattanooga Eye, Ear, Nose and Throat Society

Chairman and Secretary: Dr. C. H. Alper, 602-606 Georgia Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

Chicago Laryngological and Otological Society

President: Dr. Roland D. Russell
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 S. Michigan Avenue, Chicago 3, Ill.
Time and Place: First Monday of each month, Drake Hotel, Chicago, Ill.

Chicago Ophthalmological Society

President: Dr. Gail R. Soper
Secretary-Treasurer: Dr. Frank W. Newell, 950 E. 59th St., Chicago 37, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

Cincinnati Ophthalmologic Club

Presidium: Dr. Donald J. Lyle, Dr. Charles K. W. Ascher, Dr. Ira A. Abrahamson
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

Cincinnati Otolaryngologic Society

President: Dr. Gerson Lowenthal
Secretary-Treasurer: Dr. Edward T. Juler, 4140 Hamilton Avenue, Cincinnati 23, Ohio
Time and Place: 6:30 on the third Monday of each month except July, August, September and December. University Club.

Cleveland Ophthalmological Club

President: Dr. Carl Ellenberger
Secretary-Treasurer: Dr. Jerome A. Gans, 723 Osborn Bldg., 1020 Huron Road, Cleveland 15, Ohio
Time and Place: Second Tuesday of November, January, February and April. University Club, Cleveland, Ohio

College of Physicians of Philadelphia, Section on Ophthalmology

Chairman: Dr. George F. J. Kelly
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

Corpus Christi Eye, Ear, Nose and Throat Society

President: Dr. L. W. O. Janssen
Secretary-Treasurer: Dr. T. P. O'Brien, 309 Wilson Building, Corpus Christi, Texas
Time and Place: First Wednesday of month, September through May, Nueces Hotel

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Henry H. Gurau
Secretary-Treasurer: Dr. Harold McCoy, 616 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

Detroit Ophthalmological Club

Secretary-Treasurer: Dr. Frederick A. Lauppe, 1801 David Whitney Building, Detroit 26, Mich.
Time: Each month, November through April

Detroit Ophthalmological Society

President: Dr. Herman D. Scarney
Secretary: Dr. J. Conrad Gemeroy, 664 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, at L'Aiglon, Fisher Building

Detroit Otolaryngological Society

President: Dr. John R. Birch
Secretary-Treasurer: Dr. Bruce Proctor, 1419 David Whitney Building, Detroit, Mich.
Time and Place: Third Wednesday of each month, September through May, David Whitney House, Wayne County Medical Society Building

SECTION ON OPHTHALMOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
President: Dr. Joseph Dessoff
Secretary-Treasurer: Dr. Robert L. Norment, No. 713, 1801 Eye St. N.W., Washington, D. C.
Time: First Tuesday of November, January, March and May

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Chairman: Dr. Irvin Feldman
Secretary: Dr. John Louzan, 1801 Eye Street N. W., Washington 6, D. C.
Time and Place: 7:00 p.m., third Tuesday in October, November, January, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. John A. Cetner
Secretary-Treasurer: Dr. Frank Furlong, 713 Union Street, Schenectady, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. Keith Barnes
Secretary: Dr. William Skokan, 1708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Charles Alexander
Secretary: Dr. Ben T. Withers, 902 Hermann Professional Building, Houston 25, Texas
Time and Place: 6:30 p.m., second Tuesday of each month, October through June. River Oaks Country Club, Houston

EYE, EAR, NOSE AND THROAT SOCIETY OF HUDSON COUNTY
President: Dr. A. V. Saradarian
Secretary-Treasurer: Dr. A. J. Magee, 921 Bergen Avenue, Jersey City 6, N. J.
Time and Place: Second Monday of each month (September through May), Jersey City Medical Center

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Robert W. Dearmin
Secretary-Treasurer: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. James W. May
Secretary: Dr. Will R. Eubank, 1404 Bryant, Kansas City, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Robert W. Godwin
Secretary: Dr. Sol Ludmerer, 117 E. 8th St., Long Beach 13, Calif.
Time and Place: 6:30 p.m., third Wednesday every month

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month. September through June Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Orwyn H. Ellis
Secretary-Treasurer: Dr. Harold Owens, 1052 West Sixth Street, Los Angeles 17, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May—Otolaryngology Section.
6:00 p.m. first Thursday of each month from September through May—Ophthalmology Section.
Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

LOUISVILLE EYE AND EAR SOCIETY
President: Dr. N. H. Burkhead
Secretary-Treasurer: Dr. A. H. Keeney, Heyburn Building, Louisville 2, Ky.
Time and Place: Dinner meetings at 6:30 p.m. on the second Thursdays of alternate months, September through May. Mayflower Hotel, 425 W. Ormsby Street

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. Gerhard Straus
Secretary-Treasurer: Dr. Erwin E. Grossmann, 238 West Wisconsin Avenue, Milwaukee 3, Wisconsin
Time and Place: 6:00 p.m., fourth Tuesday of each month, October to May, University Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. J. V. V. Nicholls
Secretary: Dr. R. Charbonneau, 958 Sherbrooke St. E., Montreal, Que., Canada
Time: Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Herbert Duncan
Secretary-Treasurer: Dr. Allen Lawrence, Doctor's Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Willis Sackett Knighton
Secretary: Dr. Raymond E. Meek, 729 Park Ave., New York 21, N. Y.
Time and Place: 8 p.m., third Monday of the month, October through May, New York Academy of Medicine, 103rd St. and Fifth Ave., N. Y.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Bernard Fread
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October through May, New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. L. W. O. Janssen
Secretary: Dr. T. P. O'Brien, Wilson Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., first Wednesday of each month, October through May, Nueces Hotel

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Dick Lowry
Secretary: Dr. Clifford Blair, 1200 N. Walker, Oklahoma City, Oklahoma
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Frank J. Klabenes
Secretary-Treasurer: Dr. Frank L. Eagle, 1620 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Glen G. Gibson
Secretary: Dr. John W. Deichler, 1930 Chestnut St., Philadelphia, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY
President: Dr. Harry P. Schenck
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.
Time and Place: 8:30 p.m., first Tuesday of month October through May, College of Physicians

PITTSBURGH ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OPHTHALMOLOGICAL SOCIETY
President: Dr. Murray F. McCaslin
Secretary: Dr. C. William Weisser, 806 May Building, Pittsburgh 22, Pa.

PITTSBURGH OTOLOGICAL SOCIETY
President: Dr. Richard Hamilton
Secretary: Dr. A. J. Fischer, 623 Union Trust Building, Pittsburgh, Pa.
Time: Bimonthly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. Clifton E. Benson
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. M. K. Rothenberger
Secretary: Dr. James H. Parker, Jr., 238 N. 5th St., Reading, Pa.
Time and Place: Third Wednesday of each month, September through June (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. J. Warren Montague
Secretary-Treasurer: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. J. C. O'Neill
Secretary: Dr. J. H. Leek, 205 W. Second St., Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., second Thursday of every month, preceding the monthly St. Louis County Medical Society meeting, St. Mary's or St. Luke's Hospital, Duluth, Minn.

EAR, NOSE AND THROAT CLUB OF ST. LOUIS
Chairman: Dr. James B. Cotten
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time and Place: Third Wednesday in November, January, March and May, University Club Building

ST. LOUIS OPHTHALMOLOGICAL SOCIETY
President: Dr. Harry Rosenbaum
Secretary: Dr. Anton J. Hummel, Metropolitan Bldg., St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George H. Brunner
Secretary: Dr. Jack B. Lee, 607 New Moore Building, San Antonio, 5, Texas
Time: Second Tuesday of each month, September through May

SAN DIEGO COUNTY OPHTHALMOLOGICAL SOCIETY
President: Dr. Floyd M. Bond
Secretary-Treasurer: Dr. Oscar G. Ravin, 233 A Street, San Diego 1, Calif.
Time and Place: University Club, second Monday of each month

SAN FRANCISCO COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Max Fine, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, February, April, September and November, San Francisco **County Medical Society Building**

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. F. Hogsett
Secretary: Dr. Charles L. Gates, 305 Medical Center, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB

President: Dr. Theodore Holstein
Secretary: Dr. John Berg, 436 Medico-Dental Bldg., Sacramento 14, Calif.
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Kenneth F. Schoenrock
Secretary: Dr. L. Reed Cranmer, 316 Michigan Street, Toledo 2, Ohio.
Time and Place: Third Tuesday of each month, November through May, Toledo Club

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Harold R. Sniderman
Secretary: Dr. M. Shusterman, 345 Bloor Street West, Toronto 5, Ont., Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Joseph W. Raymond
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of April and September

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

President: Dr. Albert Abbott
Secretary: Dr. Wilson C. Marsden, 99 North Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

As of June 1, 1953

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National Model D (Duplex)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506
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Distr.: NORMATONE HEARING AID CO.
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Distr: **RADIOEAR CORP.**

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Mfr. ROCHESTER ACOUSTICAL LABORATORIES, INC.
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Mfr. TONEMASTER, INC.
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Unex Midget Models 95 & 110
Unex Models 200 & 230
Mfr. NICHOLS & CLARK
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Dallas 6

Western Electric Models 65 & 66
Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal
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Mfr. ZENITH RADIO CORP.
5801 W. Dickens Ave.
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All the accepted hearing devices have vacuum tubes. Accepted hearing aids more than five years old have been omitted from this list for brevity.

SEMI-PORTABLE HEARING AIDS

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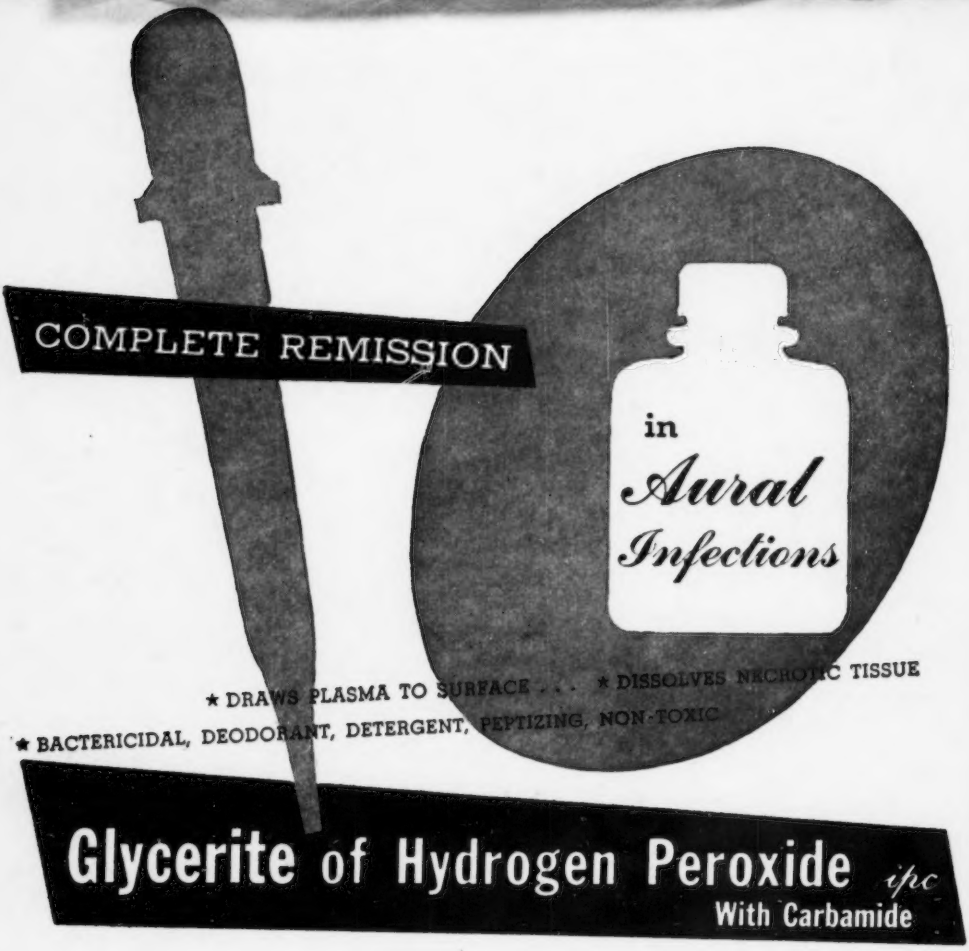
Chicago 10

Precision Table Hearing Aid
Mfr. PRECISION HEARING AIDS
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr. SONOTONE CORP.
Elmsford, N. Y.



Glycerite of Hydrogen Peroxide ipc
With Carbamide

★ BIBLIOGRAPHY

Arch. Otolaryngol., 43:605, 1946.
E., E., N., & T. Mo., 26:27, 1947.
Laryngoscope, 56:556, 1946.
New Eng. J. Med., 234:468, 1946.
Annals of Allergy, 4:33, 1946.
J. A. Ph., A., (Sc. Ed.) 35:304, 1946.

Literature on request.

Clinical studies concerned with the use of Glycerite of Hydrogen Peroxide in the treatment of chronic purulent otitis media demonstrated seventeen of twenty-nine patients in complete remission in 14 days and the remainder by the 38th day. The patients studied presented conditions existent for periods of 2 weeks to over 40 years. Previous treatment by the usual therapeutic means, including tyrothricin or penicillin, was ineffective in all cases.

Constituents:

Hydrogen Peroxide 1.446%, Urea (Carbamide) 2.554%, 8-Hydroxyquinoline 0.1%. Dissolved and stabilized in substantially anhydrous glycerol . . . q.s. ad. 30cc.

Available on prescription in one-ounce bottle with dropper.
Administration: One-half dropperful two to four times daily.

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